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ANNALS of SURGERY

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No. 2

DIABETES IN RELATION TO SURGERY

BY EDWARD WATTS SAUNDERS, M.D.

OF NEW YORK, N. Y.

FROM THE SECOND SURGICAL SERVICE (CORNELL) BELLEVUE HOSPITAL AND THE DEPARTMENT OF SURGICAL RESEARCH, CONNELL UNIVERSITY MEDICAL COLLEGE

In the diabetic patient requiring surgery, diabetes itself frequently takes a minor place in the list of the many complications which may arise. The critical and individual care received by the diabetic patient both preoperatively and post-operatively makes diabetes *per se* an asset and not a liability. If every patient suffering from diabetes had expert medical treatment and could or would live under improved hygienic conditions, infection would not occur any more often than in the non-diabetic patient. This is proven by the fact that there are at least 100 public ward diabetic patients requiring surgery to 1 private, well-cared-for diabetic patient.

The child or young adult diabetic is rarely seen on a surgical service, the disease having manifested itself sufficiently to require medical attention. The older diabetic has either had the disease unknowingly or has been able to carry on perfectly well without treatment. This undoubtedly is the reason for the many complications which are present when infection finally intervenes and surgery is necessary.

The prognosis is about proportional to the duration of the disease, either known or unknown, in an individual. If prophylaxis is to be encouraged, a routine blood-sugar determination should be as important as a routine Wassermann for the blood and tissue sugar may be considerably higher than normal without spilling over in the urine. The patient who has had a high fat and low carbohydrate diet over a period of years undoubtedly develops arteriosclerosis much more rapidly than the patient on a well-balanced diet. The resulting disturbed fat metabolism, cholesteramia, cholesterol deposits on the intima of the blood-vessels of heart, kidney, and extremities, are the basic factors that cannot be changed when infection intervenes. Acidosis, dehydration, malnutrition, high blood and resulting high tissue sugar, optimum acid pⁿ of tissue favoring glycophilic organisms in their growth and toxin production, can all be corrected in a comparatively short period.

The improvement, therefore, of surgery in relation to diabetes lies not in surgical technic but in the prophylactic prevention of cholesteræmia in the patient suffering from diabetes. These statements are well substantiated in the following review of eighty diabetic patients requiring surgical intervention who had either been completely innocent of their diabetic state, or who had known but were untreated, or who had known and were treated by a

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high fat, low carbohydrate diet. Extremely few had ever been treated with insulin and then only sporadically.

Patients Su Acidosis and Never treat Positive blo High N.P.J Albumin an Hypertensio	d dehyd ed for od Was N. (35- d casts	ration production production production production rational production produc	n						26 5 20
Blood sugar	rs		U	pon ad	mission			Upon o	lischarge
Average blood s	ugar		146 mg.	per 100 cc.					
		N	Tumber	of Case	es, 1916	-1031			
Age	20-30				60-70			Mortality	Mortality per cent.
Before insulin*	1	0	4	10	9	0	24	15	62.5
Since insulin†	0	3	13	28	10	1	55	14	25.45
* Before insuffice insuffi				1931.					
			Opera	ative P	rocedur	es			
Amputation . Carbuncle—drain Conservative—di metatarsal n No operation Hand infection—Acute appendicit Amputation brea	git and emoval -incision	1		20	Carcin Meser Acute Intest Perin Osteom Fracture Hæmor	noma— nteric t choled inal ob ephritic yelitis e	bowel a hrombo cystitis struction absces	and pelvic	
* 6 died, 24	per cer	nt.							80

Pre-operatively the attempt has been to correct acidosis and dehydration with glucose and fluid by mouth, vein, and clysis covered by insulin, I unit per gram as a rule. If these two factors are not corrected, the surgical risk is increased considerably. This holds just as true in a carbuncle as in an apparently more serious infection. The blood sugar is reduced by getting rid of the infection. Operation is, therefore, indicated, immediately upon the elimination of an existing acidosis or immediately if acidosis is not present. Incision should always extend into healthy tissue to eliminate the possibility of extension.

Post-operatively an attempt is made to keep the carbohydrate intake around 250 grams in twenty-four hours. This can be done in any case by mouth, infusion, and clysis. Usually I unit of insulin per 2 grams of sugar is administered with each intake. Four-hourly urine specimens are tested, and a graded dose of insulin, from 40 to 10 units for a 4 plus to I plus Benedict's reaction, is administered. The surprising thing is the rapidity with which the blood sugar falls and the insulin requirement drops as soon

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as the infection is incised or excised. A blood sugar of 500 milligrams per 100 cubic centimetres or more and an insulin requirement of 150 units in twenty-four hours will drop to normal in a period of twelve hours. If it does not, the infection is not surgically removed or there is a blind infection present. This holds true in every case except for the infections due to non-toxin-producing organisms. Two cases illustrate this point very clearly.

An obese woman aged thirty-six with a rapidly extending cellulitis of the right foot and leg, blood sugar 600 milligrams per 100 cubic centimetres, extreme acidosis and impending coma was treated conservatively (multiple incisions) because of her normal circulation. A beta streptococcus was cultured. The temperature ranged between 101° and 104° for four days. The infection continued to spread so a mid-thigh amputation was performed. The blood sugar and insulin requirement dropped, but in a few days began to rise although the stump remained uninfected. The insulin requirement continued to ascend until 195 units were required in twenty-four hours. She then began to complain of pain in the right upper quadrant. A mass could be palpated, a cholecystostomy was done and a huge empyema of the gall-bladder drained. A peripancreatitis or cedema of the pancreas was present. The blood sugar immediately dropped and within four days the insulin requirement was five units three times a day on a 250 carbohydrate intake.

An elderly patient aged sixty-five showed signs and symptoms of acute cholecystitis with probably peripancreatitis. She was definitely jaundiced, blood sugar 363 milligrams per 100 cubic centimetres, extreme acidosis, and approaching coma. Vigorous glucose and insulin therapy immediately cleared up her acidosis. She improved so rapidly that a cholecystostomy was not done. Within four days the blood sugar was normal, jaundice had completely disappeared and no insulin was required. Without any rise in the blood sugar a fluctuant non-tender buttock abscess developed which was drained. A staphylococcus was cultured. The abscess drained and healed without any appearance of sugar in the urine or necessity for insulin. The patient returned to the hospital two months after discharge complaining of pain in the right flank of three weeks' duration. A tender, soft mass had formed in that region. The blood sugar was 94 milligrams per 100 cubic centimetres and there was no sugar in the urine. A perinephritic abscess was drained, staphylococcus aureus cultured, and the patient healed her abscess rapidly without a rise in blood sugar. The patient continued her convalescence on a regular diet without insulin.

It seems logical to assume that infections due to avirulent non-toxinproducing organisms do not change the diabetic state with the one exception of inflammatory lesions about the pancreas, namely those involving the gallbladder, stomach, and duodenum. The importance of not giving insulin unguarded by glucose pre-operatively cannot be too greatly stressed for a patient may well go into shock while under the anæsthetic if the diabetic state is not severe.

In spite of the fact that the hypoglycæmic state is a precarious one as Root has emphasized, it is the one possibility of increasing conservative operations. In five patients in this series, conservative incision and drainage of the extremity was successful. Their blood sugars were maintained between 80 and 100 milligrams per 100 cubic centimetres until the infection subsided. This is impossible, however, unless the surgical procedure has stopped extension of and absorption from the infection. Their carbohydrate

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intake was 250 grams. The patient does not feel well nor does he gain weight, but the infection subsides much more rapidly. With subsidence of the infection, the blood sugar is allowed to rise and the maximum carbohydrate intake maintained. More conservative operations could be accomplished of the type which McKittrick so ably describes if the tissue hyperglycæmia and optimum p^H for toxin formation could be immediately corrected. The infecting organism is invariably glycophylic, toxin is formed only at a very narrow p^H range, both of which seem to explain why an infection in diabetic tissue extends at such a rapid rate. What actually happens, whether toxin stimulates adrenalin secretion which fixes insulin, or whether it is the direct action of toxin on the pancreas, is immaterial to our present discussion. The enhanced growth of the organism is the important factor.

Pillsbury has shown that a specific carbohydrate metabolism of the skin may be observed experimentally and that in the presence of dextrose in the incubating medium, lactic acid formation is increased. If true, this would prove both factors, increased dextrose and optimum $p^{\rm H}$ favoring growth and toxin formation.

Correcting acidosis and dehydration, maintaining a low blood sugar with resultant low tissue sugar, giving a high carbohydrate intake (250 grams or more) aid tremendously when conservative surgery or no surgery is attempted. A warning is necessary, however, against glucose infusions and glucose clyses in the presence of transient bacteræmia unless enough insulin is given to burn the injected glucose immediately. Glucose by vein in any bacteræmia causes the colony count per plate to increase tremendously and an abscess may develop at the site of a glucose clysis.

The choice of operative procedure is extremely well discussed in McKittrick's book of diabetic surgery. Where the patient's chances are slight and amputation seems necessary, the least shocking procedure should be performed. A Stokes-Gritti is then the type of choice. Otherwise the patient's economic situation should determine the site of amputation. It is our feeling that general anæsthesia is as safe as spinal anæsthesia.

Careful care of the patient cannot be too greatly stressed and is well illustrated by two cases. One patient who had a fracture of the neck of the femur developed a carbuncle of the back beneath the cast because of pressure and position. It extended so rapidly that incision and drainage was not performed soon enough to save the patient.

Another with cellulitis of the palm of the hand, incised and drained and watched carefully for two weeks, was discharged to be dressed in the outpatient department with wounds still draining. Care necessarily was not so strict and within a week the patient had developed a hæmolytic streptococcus in the wound which rapidly extended throughout the arm. Bacteræmia resulted and the patient died in three days. Since the patient can never be trusted to carry out orders which seem unnecessary to him, he should not be discharged until the wounds are healed sufficiently to prevent re-infection.

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The inciting organism or organisms are the most important factor. Multiple organisms are more difficult to control than a single infecting organism. Staphylococci are the easiest to control. Streptococci produce necrosis and toxin and spread rapidly. Streptococcus gangrene is hopeless in the diabetic unless treated within a few hours following the onset of the infection. The only proven case bacteriologically in this group began in the foot and had extended to the muscles and fascia of the abdominal wall twenty-four hours later. Anaërobic bacilli of the gas-producing type are usually at the site of amputation and the stump becomes infected. The possibility of this complication, when recognized, will be much less with a wound left entirely open than with a closed wound favoring anaërobic growth.

The differential diagnosis between impending coma and acute surgical abdomen has occurred only twice in this series.

An obese patient aged forty complained of sudden acute pain in the abdomen. The blood sugar was 400 milligrams per 100 cubic centimetres. In spite of the most vigorous glucose-insulin therapy, the patient died within twenty-four hours, the blood sugar mounting to 700 milligrams per 100 cubic centimetres. Although an autopsy was not granted, it was felt that the patient had an acute pancreatitis secondary to gall-bladder or stomach inflammation.

A correlation between gastric and duodenal ulcer and diabetes has been made several times. One case following a gastroenterostomy for a posterior duodenal ulcer with considerable inflammation on the surface of the pancreas had to have vigorous glucose-insulin therapy post-operatively for five days before the seemingly transient glycosuria was under control. The consensus of opinion that impending coma gives symptoms of an acute surgical abdomen may, therefore, in each case be due to a peripancreatitis secondary to gall-bladder or stomach disease. Certainly the diabetic patient over the age of forty does not lose carbohydrate control without infection somewhere. The resulting peripancreatitis could well explain the transient diabetes. Joslin reports nineteen diabetic patients with duodenal ulcer and nine with gastric ulcer. Jankelson reports six with duodenal ulcer.

Another patient, aged sixty-six, complained of a sudden severe attack of pain in the abdomen which became steadily worse. The diabetes was easily controlled. At operation, three feet of gangrenous small intestine were found and resected. The patient did well until the seventeenth post-operative day when she had a sudden severe pain in the chest and dyspnæa and died four hours later in spite of all medication. At autopsy many emboli were found in the lungs and the remainder of the small intestine had become gangrenous from embolic occlusion of vessels.

The burden of proof, therefore, lies with the one who says impending coma can give symptoms identical with those of acute inflammation in the abdominal cavity, for both peripancreatitis and subacute pancreatitis may subside without surgical interference. Also gradual sclerosis of the mesenteric vessels may cause abdominal pain without causing loss of viability from mesenteric thrombosis as in the case cited above. If sudden acute infection

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about the pancreas can cause a mild diabetic state to become a severe diabetic state and a non-diabetic state to become a transient diabetic state (glycosuria), it would seem logical to raise the question whether the true diabetic state may have resulted from chronic persistent infection of the pancreas by the type of streptococcus found in the gastro-duodenal area; the characteristic feature of this infective agent is that it does not produce the recognized picture of an acute or chronic inflammatory reaction in the involved tissue.

CONCLUSIONS

I. The severity of infections in patients suffering from diabetes is probably due to the enhanced growth of glycophilic organisms in tissue abnormally high in glucose and with a p^H most favorable to toxin production.

2. Ninety per cent. of the diabetic patients requiring surgery are over fifty years of age and are suffering from complications much more severe than diabetes.

3. Maintaining a low blood sugar with resultant low tissue sugar and change of tissue p^H helps considerably in controlling the existing infection when conservative surgery is attempted.

4. Gastric or duodenal ulcer, gastritis or duodenitis, or acute cholecystitis may cause cedema of the pancreas, peripancreatitis or subacute pancreatitis which, by causing an exacerbation of the diabetes as well as by causing an actual acute surgical condition, might well explain the difficulty in the differential diagnosis between impending coma and acute surgical abdomen.

BIBLIOGRAPHY

- Joslin, Elliot P.: The Treatment of Diabetes Mellitus. Lea and Febiger, Philadelphia.
 McKittrick, Leland S., and Root, Howard F.: Surgical Diabetes. Lea and Febiger, Philadelphia.
- ^a Root, Howard F.: Diabetic Gangrene. Archives of Surgery, vol. xxii, February, 1931.
 ⁴ Jankelson, I. R., and Rudy, A.: The Simultaneous Occurrence of Peptic Ulcer and Diabetes or Glycosuria. The American Journal of Medical Sciences, vol. clxxxi, March, 1931.
- ⁵ Pillsbury, Donald M.: The Intrinsic Carbohydrate Metabolism of the Skin. J. A. M. A., vol. xcvi, February 7, 1931.

MORTALITY IN GOITRE OPERATIONS*

AN ANALYSIS OF A SERIES OF 1,755 OPERATIONS DURING THE TEN-YEAR PERIOD, 1920-1929 INC.

BY EMIL GOETSCH, M.D. OF BROOKLYN N. Y.

FROM THE SURGICAL DEPARTMENT OF THE LONG ISLAND COLLEGE HOSPITAL

It is my purpose in this report to analyze the factors responsible for the fatal outcome in twenty-two instances of operations for goitre in a series of 1.755 operations carried out during the ten-year period from January 1, 1920, to January 1, 1930. A frank discussion of our failures rather than our successes is a wholesome lesson and one not sufficiently indulged in. For convenience in this study, this ten-year period was chosen, as it covers the first ten years' work of the thyroid clinic at the Long Island College Hospital. During the earlier years the operations for goitre were done by myself and during the latter years I was joined in the operative work by Dr. Arthur Goetsch. It is desirable to mention at once that this operative work covers a period approximately five years before and five years after the institution of the standardized pre-operative intensive preparation of thyroid patients with iodine. Certain comparisons can be made between the results in the early operative cases and in the latter ones, as influenced by this therapeutic procedure. This point will be discussed later and the influence of iodine will be commented on more fully.

In the study of twenty-two fatalities in this series, the cases may be subdivided as to cause of death into several convenient groups. They may also be grouped according to diagnosis and according to the type of operation performed. However, these are not the important factors and it will therefore be more interesting and profitable to discuss the clinical factors of danger in thyroid surgery. This study is aided by the fact that certain factors have remained rather constant. The operative technical procedures have become standardized. The pre-operative preparation, except for the element of iodine therapy, remained practically the same. The anæsthetic used was almost entirely gas-oxygen with occasional small amounts of ether. Ethylene was used for a time. Local anæsthesia was used in most of the ligations. The deaths, therefore, were not primarily dependent upon differences in operative technic, preparation of the patient, or anæsthesia, and we can therefore speak of dangerous factors other than those concerned primarily with the operation itself. The post-operative care has also become practically standardized.

Report of Fatalities.—In this series of 1,755 operations there were twenty-two fatalities; sixteen were in instances of exophthalmic goitre and six in instances of toxic adenomatous goitre. There is an operative mortality thus of 1.25 per cent. covering all types of operations and all degrees of pre-

^{*} Read before the New York Surgical Society, February 25, 1931.

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operative toxicity and general clinical severity. There was a total of 1,553 patients of all types of goitre. Operative mortality as to patients is therefore 1.41 per cent. One must remember that this includes many patients before the time that we were able to operate as safely as we can now as a consequence of the intensive pre-operative iodine treatment which was introduced six to seven years ago and which has rendered operation in toxic goitre much more safe. It is interesting that in our experience the mortality rate was no greater in the days before the iodine treatment than it has been since. Before the introduction of iodine, we gauged very carefully our operative procedure and the toxicity of the case, and as a consequence, there were many more ligations and partial resections than there have been since the introduction of iodine. Following the general use of intensive iodine treatment immediately before operation, bilateral resection operations have become the rule and ligation and partial resections are comparatively rare. However, as will be shown later, some of the principal difficulties in treating the thyroid patient, since the introduction of the pre-operative iodine treatment, have unfortunately been the dangers introduced by the incorrect use of iodine. These dangers have contributed to the mortality in several instances.

Of twenty-two deaths in a series of 1,755 operations on 1,553 patients, sixteen were in instances of exophthalmic goitre as follows:

Type of operation for exophthalmic goitre	Deaths	Previous preparatory operations
Ligation of single artery	4	Cases 1, 2, 3, 4-None
Ligation of two arteries	2	Case I—Ligation both superior thyroid arteries
		Case 2-Resection attempted
Resection unilateral	4	Case I—Ligation one superior and both inferior thyroid arteries
		Case 2—Ligation both superior thyroid arteries
		Case 3—Two partial resections of right lobe (done elsewhere)
		Case 4—Ligation one superior thyroid artery (done elsewhere)
Resection bilateral	6	Cases 1, 2, 3, 4-None
		Case 5—Ligation of right and left su- perior thyroid arteries (two stages)
		Case 6—Ligation both superior thyroid arteries
Total	16	

Six deaths were in instances of adenomatous goitre as follows:

Type of operation for adenomatous goitre	Deaths	Previous preparatory operations
Resection unilateral	2	Case 1—None
		Case 2—Two partial unilateral resec- tions (done elsewhere)
Resection bilateral	4	Cases 1, 2, 3, 4-None
Total	6	

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It is clearly impossible because of the limited time to discuss in any detail whatever the individual fatalities occurring during this ten-year interval. This will be reserved for a subsequent report. At the present moment we can simply give a brief summary and discussion of the factors which were either directly or indirectly responsible for the fatal outcome in the series of operations here reported.

SUMMARY AND DISCUSSION OF THE FACTORS RESPONSIBLE FOR THE TWENTY-TWO FATALITIES

The causes of death in a series of 1,755 operations for goitre on 1,553 patients during a ten-year period in the order of their importance as indicated by a study of the twenty-two deaths which occurred, are first:

Post-operative Hyperthyroidism with Secondary Cardiac Failure.—Eight of the twenty-two deaths, or 36 per cent., occurred as a result of post-operative hyperthyroidism with secondary cardiac failure. The cause of death in this group is to be distinguised from that due to primary cardiac failure (unassociated with post-operative hyperthyroid crisis) and resulting from severe myocardial damage and deterioration from prolonged hyperthyroidism. Six of the eight fatalities occurred in instances of exophthalmic goitre and two followed resection of toxic adenomatous goitre. As to preliminary-stage operations, the six exophthalmic goitre cases group themselves as follows: Death followed in one instance after ligation of the right superior thyroid artery only; in one instance in a patient who had a right hemithyroidectomy after two preliminary ligation operations; in two instances after a bilateral resection in which there had been no preliminary operation; and finally in two instances of bilateral resection preceded by a preliminary ligation of both superior thyroid arteries.

It is seen then that, to a very definite extent, the fatal outcome is not directly related to the type of operation performed, for fatalities occurred after ligation, unilateral resection and bilateral resection operations. The factor of post-operative hyperthyroidism is, to be sure, primarily related to the degree of pre-operative hyperthyroidism which, in turn, had often been unfavorably influenced by the indiscriminate use of iodine, with resultant exacerbation of the symptoms. On the other hand, after prolonged use of iodine, particularly in small doses, the patient may appear reasonably calm, the pulse may be only moderately accelerated, and the basal metabolic rate may or may not be so high as to act as a warning. The general condition of the patient and the metabolic rate are relatively little influenced in such instances by pre-operative intensive treatment with iodine, since the thyroid gland, at such times, is refractive to further iodine medication. As a consequence, the surgeon may obtain a feeling of false security and be surprised to witness a severe post-operative reaction. Such iodine exacerbation is difficult to control. The usual measures of rest, further iodine treatment, or even ligation, are of limited use.

The bad results of the indiscriminate use of iodine were noted in seven

of the eight fatalities in this group and I have not the slightest doubt that the incorrect use of iodine was a definite factor in causing the fatal outcome. The one fatality following ligation, in a patient who had had no iodine treatment, could have been avoided, I feel certain, by the pre-operative administration of iodine. This death occurred, however, in 1920 before the pre-operative iodine treatment had been generally introduced.

I believe our experience justifies us in saying that, by and large, iodine has no place in the medical treatment of exophthalmic or adenomatous goitre. It should not be used. It deprives the patient and the surgeon of the protective action inherent in iodine when given intensively as a pre-operative measure. In many instances iodine that is given presumably to effect a medical cure merely converts the inactive case into an active one, and the active case into a desperate one and thus renders the operative procedure exceedingly dangerous. Consequently, regardless of whether a ligation, a single lobectomy or double resection is performed and regardless also of whether the intensive pre-operative treatment with iodine is or is not employed after the thyroid gland has become refractive, the operation is commonly fraught with danger. The safest procedure is to give the patient a period of sedative treatment for two to six months or longer depending upon the circumstances. Iodine can then again be given with satisfactory benefit and the operation again becomes relatively safe. In short, iodine should be employed solely to prepare the hyperthyroid patient for operation.

The value of basal metabolic rate determinations in warning the operator as to when he may expect a severe post-operative hyperthyroid reaction should be considered particularly when we are dealing with the patient previously treated with iodine. In the untreated case it is a common experience that the basal metabolism gives us the most helpful information. We can judge rather accurately as to the best time to operate and as to the extent of the operative procedure best suited to the patient. On the other hand it seems from our study that when a patient has been indiscriminately treated with iodine the danger signals are often obscured and the outward calm of the patient may give the operator a feeling of false security. It is our feeling that the basal metabolic rate of itself is under such circumstances of less value in helping us to decide as to the operability of the patient. Thus, of the eight patients succumbing to primary hyperthyroidism and secondary cardiac failure, seven patients had received prolonged treatment with iodine. The basal metabolic rates varied between plus 47 and plus 72 (not excessively high). Notwithstanding the favorable basal metabolic rates, fatalities ensued regardless of whether a ligation, partial resection or double resection had been performed and furthermore regardless of whether additional iodine had or had not been administered intensively before operation. I wish to stress that pre-operative iodine treatment does not protect the iodized patient in any way comparable to the protection afforded the patient not previously so treated. It is our definite opinion that some of these fatalities would not have occurred had iodine not been previously given. A high metabolic rate

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in a patient previously iodized may either not fall at all or decrease only slightly after further intensive treatment.

Primary Cardiac Failure.—Four fatalities occurred after operation for exophthalmic goitre on patients in the terminal stage of the disease. These patients had suffered with toxic goitre producing active hyperthyroidism over periods of three, five, fifteen and twenty-nine years respectively. It is interesting that there were no cases of adenomatous goitre in this group.

The principal pre-operative manifestations in all these patients were referable to a badly damaged heart, which showed, in the severer cases, a complete breakdown, with dilatation, fibrillation, complete decompensation resulting in pulmonary congestion, cyanosis, ascites and general anasarca. Three of these cases had had indiscriminate iodine therapy, which had been given in one case by means of injections into the goitre. One in this group of four cases upon whom ligation was performed had received no iodine either medically or as a pre-operative measure. In the only instance where bilateral resection was performed, further iodine was given as a pre-operative measure as well. The type of operation performed was thus not the determining factor in the fatal outcome for the operations performed were a single ligation in one case, ligation of two arteries in two instances, and one bilateral resection. Twice the hyperthyroidism was fairly severe at the time of operation and in two cases it was not. Only once was the post-operative reaction considered in any way contributory to the fatal outcome. Deaths occurred as follows: One in forty-eight hours (bilateral resection-preoperative intensive iodine given) (indiscriminate treatment); two in five days and one in five months following operation.

These fatalities are therefore not considered to be due primarily to the factor of iodine therapy, to the type of operation performed or to the degree of hyperthyroidism present at the time of operation. They were referable solely to the status of the cardio-circulatory system. The heart in each case was too badly damaged and had gone beyond the limits of benefits to be derived from any type of operative procedure. There was no power of recuperation on the part of either the heart or the generally damaged patient. One should have refused to operate at all in these instances and not attempt the impossible. Whether treated medically or by operation, the result in either event would have been disastrous.

Pneumonia.—Of the twenty-two deaths there were three resulting from pneumonia, two of the lobar and one of the bronchial type. Two followed bilateral resections for adenomatous goitre and one followed a right-sided resection for exophthalmic goitre. Medical treatment with iodine had been given except in one of the cases of adenomatous goitre. The degree of hyperthyroidism present before operation was not a factor in the fatal outcome and the basal metabolic rate was not high in any of the three cases. Uniformly in these patients who died of pneumonia, we find a bad previous history, referable to the cardio-respiratory system. Thus in the first instance (M. L.), a large adenomatous goitre had produced serious respiratory diffi-

culty for four years. Four months preceding operation she had had attacks of acute suffocation, which had placed a great strain on her heart and lungs. Finally, operation had to be done to prevent death from acute suffocation. She died of bronchopneumonia on the evening of the third day following operation. The prolonged acute tracheal obstruction with cardiac and pulmonary embarrassment, tracheitis and general exhaustion were the predisposing factors causing the fatal pneumonia.

The second fatality, due to pneumonia, followed a right-sided resection in exophthalmic goitre. This patient was in a state of fairly acute iodine exacerbation. He was extremely weak and had lost a great deal of weight. His past history was bad. He was a sufferer with chronic bronchitis and tuberculosis and had had a fairly recent pulmonary infection. He was an utterly bad operative risk. Every pre-operative measure calculated to protect this patient had been given. He died two days after operation of an extensive fulminating pneumonic process. Operative relief should not have been attempted.

The third death of pneumonia (A. J.) followed upon a double resection for a moderately toxic adenomatous goitre in a woman who had a history of a previous bronchopneumonia, arteriosclerosis with hemiplegia which subsequently cleared up, hypertension, a moderately severe diabetes, and myocardial insufficiency. She was also an utterly bad risk. Pre-operative iodine had been administered. The immediate post-operative course was not alarming. However, in several days, lobar pneumonia developed and she died five days after operation.

We might be criticized as exercising bad judgment in operating at all in two of these cases. However, I may say that only in the rarest instances did we refuse to offer such benefits as operation might yield. In short, we did not discriminate against any patient who offered even a slight hope of improvement. It is readily seen that in the three instances of post-operative pneumonia there was a history of more or less serious cardio-pulmonary complications together with other systemic disorders and general exhaustion. In one case there had been prolonged tracheal obstruction. It is needless to point out the possible dangers following operations on patients presenting such conditions. Considering that there were only three deaths from pneumonia in 1,755 operations, a percentage of less than 0.2 per cent. (actually 0.17 per cent.) and that in the series there were patients ranging in age from five to seventy-nine years, with all degrees of hyperthyroidism and general debility, and that many were practically in the terminal stages of the disease, we must regard ourselves fortunate. I attribute this satisfactory result to the precautions of never operating in the presence of a "cold" or bronchitis or when there was a history of recent respiratory infection; of using a light gas-oxygen or ethylene anæsthesia which allows early recovery of the throat reflexes, thus avoiding aspiration; of cutting short the duration of the operation, avoiding unnecessary loss of blood, trauma, and respiratory embarrassment. It should be emphasized also that the greatest

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care was exercised in avoiding exposure and even the slightest trauma of the recurrent laryngeal nerves, in order to prevent the possibility of aspiration pneumonia which might result from interference with the reflexes of the larynx. Tracheal compression during the operation was carefully avoided since prolonged respiratory embarrassment with cyanosis obviously predisposes to pulmonary complications.

Embolism.—There were three patients, all women, aged twenty-seven, forty-seven, and forty-eight respectively, who died as a result of post-operative embolism. They were all instances of active exophthalmic goitre. In one no iodine therapy had been given. The basal metabolic rate in this case was plus 63. In the remaining two cases, indiscriminate iodine treatment had been given and in both there was a definite exacerbation; in one of these the basal metabolic rate was quite high, namely plus 68. One (M. S.) death followed ten days after the ligation of only one artery; one occurred nine days after the ligation of one artery and one followed twelve days after a right-sided resection. The extent of the operation played no rôle in the fatal outcome, since in two instances the least that could be accomplished surgically, namely, the ligation of a single artery, had been done. In all of the three cases of embolism, there had been, however, very serious cardiac damage produced by the preceding very marked hyperthyroidism.

In the first case, there was in addition a history of rheumatism. A diagnosis of chronic endocarditis had been made in addition to exophthalmic goitre. The heart showed a loud systolic murmur at the left border of the sternum. The pulse was 130, and the basal metabolic rate plus 63. The convalescence was satisfactory until the sixth day when she developed symptoms and signs of cerebral embolism and thrombosis and died on the tenth day following a ligation operation. The final signs were a right facial paralysis and right hemi-paresis.

In the second case, cardiac symptoms had been predominant throughout her period of illness which lasted a year and a half. Iodine treatment had been given and had produced a phase of extreme exacerbation. The cardiac action was forceful, with frequent extrasystoles and there had been long periods of fibrillation. After general treatment and rest the basal metabolic rate was found to be plus 45. After a single ligation operation and a further period of rest and general treatment lasting four months she had made a definite improvement. The irregularity of the heart with fibrillation had, however, continued. Finally a right-sided resection was done. Precordial pain appeared twenty-four hours after operation. On the fifth day she suffered severe pain over the precordium and in the left leg below the knee. The leg became cyanotic and the pulse in the larger vessels disappeared. On the seventh day the heart was fibrillating markedly, and had become definitely enlarged. On the eleventh day, embolism occurred in the right leg, gangrene subsequently developed and the patient died in coma on the twelfth day following operation. The post-operative reaction of itself was thus no factor in the final outcome.

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In the third case, a young woman of twenty-seven, a marked hyperthyroidism had existed for two years. The basal metabolic rate was plus 68.

She was extremely active. A thrill and a systolic blow, due, we thought, to
an old chronic endocarditis, were heard over the apex of the heart. The
pulse was 145. In this instance there were no cardiac irregularities, cedema
or ascites. On account of the unusual toxicity, the simplest surgical procedure was chosen, namely, the ligation of a single thyroid artery. One
week after operation the condition was so satisfactory that the patient was
told she might soon go home. Early on the morning of the ninth day, while
eating her breakfast, she suddenly became weak, pale and speechless and in
a few moments expired. Our conclusion was that she had had a coronary or
pulmonary embolism on the basis of an old endocarditic lesion. This is the
only death in the series the cause of which was at all doubtful and postmortem examination was unfortunately not obtained.

In these three fatal cases of embolism, the symptoms and signs of severe cardiac damage were apparent. Two were complicated by an old endocarditic lesion. In all cases in which the hyperthyroidism has existed for a long time, and in which there is extensive cardiac damage with dilatation and prolonged periods of fibrillation and particularly when all this is associated with a history of rheumatism and possible endocarditis we should keep in mind the possibility of embolism. The infrequency of thrombosis and embolism in this series of goitre operations seems remarkable when we consider that the operations are performed in a very vascular field and that the old adenomatous and intrathoracic goitres are particularly prone to develop large venous plexuses. I have wondered why this should be and I believe that the rapidity of the blood-stream produced by the tachycardia is the important matter. We know that slowing of the blood-stream is one of the principal factors in the etiology of thrombosis and subsequent embolism.

Tetany.—One patient in our series of fatalities died of tetany. She had an exophthalmic goitre producing severe hyperthyroidism for a year. The gland had enlarged irregularly, especially posteriorly, and on account of adhesions, delivery of the lobes was more than ordinarily difficult and some force had to be used in raising them. This difficulty in mobilization of the gland had doubtless caused injury to the parathyroid glands for on the day following operation symptoms and signs of mild tetany such as drawing sensations in the fingers and forearms and a slightly positive Chvostek's sign appeared. Parathormone was administered intravenously and calcium lactate was given by mouth. On the ninth day the symptoms suddenly became more severe. Parathormone was given in accordance with indications. Symptoms disappeared and the patient was thought to be doing well. Since this was the only case of severe tetany in our series, the staff was not entirely familiar with such complications and was doubtless off its guard for on the eleventh day after operation she suddenly was seized with a major attack. She died during this final acute typical spasm even before parathormone could be given.

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Tetany in this case was due we believe to trauma of the parathyroids, with temporary suspension of their function and not to their removal, inasmuch as the usual type of resection had been carried out and the patient lived considerably longer than would have been possible had the parathyroid glands been removed. It is naturally not necessary to remove the parathyroid glands, to cause the characteristic tetany. Simple injury may cause failure to function. The slightest injury at the proper point may cause thrombosis of the very minute artery of supply, thereby suspending glandular function for the time being. I wish to emphasize further, that whenever there are even the mildest symptoms of tetany and a positive Chvostek's sign, parathormone should be administered daily and its continued use should be controlled by blood-calcium determinations. It should gradually be suspended when the symptoms and signs have entirely disappeared.

When the parathyroid bodies have been traumatized, they act in the nature of transplants. Injections of parathormone must be continued until the circulation of the parathyroids has become re-established and there has been a return of secretory activity which may require from ten or twelve days to two or three weeks or even longer. I believe that during the transient intervals of entire freedom of symptoms in our fatal case, the staff had not considered the patient on the threshold of more severe spasm, had allowed the judicious administration of parathormone to lapse and had thereby permitted the occurrence of a severe tetanic spasm with respiratory failure. If daily administration of the necessary amount of parathormone had been given, and if this had been controlled by frequent blood-calcium determinations and careful observation of symptoms and signs, this patient would not have died. Great care must be exercised during operation on the thyroid gland to avoid injury to the parathyroid bodies in order to prevent a similar complication. The danger of complete removal need hardly be stressed for this would be a purely technical error, which should not occur in the present state of our knowledge of the resection operation. It is a satisfaction that this distressing complication occurred in only one instance.

Tracheal Obstruction—Respiratory Failure.—There was one fatality due to respiratory failure caused by tracheal obstruction and laryngeal cedema in a large stout woman who had had a large adenomatous goitre for twelve years. Her neck was very short and fat, a fact which increased somewhat the operative difficulties. A bilateral resection was done. On the following day considerable cedema of the neck was noticed and after forty-eight hours there was some difficulty with respiration. Dyspncea and cyanosis appeared, the respiratory obstruction increased and her condition became critical. An immediate tracheotomy was done at the bedside, but death occurred suddenly as a result of respiratory failure.

There was doubtless softening of the tracheal rings, the result of compression by the goitre for a period of twelve years or more. The ædema of the entire neck and of the tracheal and laryngeal mucosa, together with the possible collapse of the trachea, had produced prolonged respiratory difficulty

and finally acute obstruction with failure of the respiratory mechanism. It is possible that an earlier tracheotomy would have saved the situation. The factors of post-operative hyperthyroidism or cardiac failure naturally do not come into play in this instance. This was the only case in the entire series of operations in which it was necessary to perform a tracheotomy for relief of post-operative respiratory embarrassment.

Accidental Death Following Intravenous Injection of Commercial Dextrose U.S.U. 5 per cent. Solution.—A patient aged forty-five years with an adenomatous goitre which had been partly resected elsewhere, on two previous occasions, was operated upon, a partial resection of the left lobe being done. On the day following operation, because of some dryness of the tongue, restlessness and irritability and to increase the fluid intake, an intravenous injection of 350 cubic centimetres of 5 per cent. dextrose (Merck U.S.P.) solution was given. This was followed by a chill and restlessness. A more severe chill followed. The pulse and respirations became rapid, the patient lapsed into a semi-coma and in half an hour the respirations ceased. The heart continued to beat for about five minutes longer. Death was considered to be due to respiratory failure. Upon investigation it was found that dextrose U.S.P. had by mistake been used instead of dextrose C.P. and on account of the increased acidity of the former the hydrogen-ion concentration of the blood had doubtless been disturbed with consequent respiratory paralysis. Dextrose administration had proven to be of such decided benefit in the post-operative treatment of our patients suffering with acute toxæmia, that we had resorted to it ad lib. Post-operative restlessness, nausea and vomiting and acetonuria are almost immediately relieved by it. Since the introduction of the pre-operative iodine therapy, the post-operative period is usually calm and glucose solutions are only occasionally given intravenously. Properly buffered solutions of dextrose are now made up or may be procured from reliable drug houses. These may be used without practically any subsequent reaction. Needless to say, the dextrose should always be of the C.P. quality.

Wound Infection (Streptococcus) Terminal Bronchopneumonia.—In only one instance in the twenty-two fatalities wound infection with streptococcus played a serious rôle. This followed operation for an active exophthalmic goitre in a young man aged twenty-eight. On the evening of the third day after he had recovered from a rather sharp post-operative reaction, the temperature began to rise and on the fourth day it reached 104°. The wound was opened and was found to be extensively infected with the streptococcus. There was considerable tissue necrosis. The patient became acutely ill. He developed a bronchopneumonia and died after a brief period of coma.

Death in this case must be considered as due primarily to a very severe wound infection and secondarily to terminal bronchopneumonia. After forty-eight hours, when we regarded the patient out of danger, the secondary acute infection led to an overwhelming toxæmia with bronchial infection, coma and death. It is probable that had we suspected the wound infection

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earlier, opened it widely on the day following operation and subjected it to generous irrigation, the patient would have survived.

CONCLUSIONS

There were twenty-two deaths following 1,755 operations on 1,553 patients, an operative mortality therefore of 1.2 per cent. An analysis of these twenty-two fatalities shows that the causes of death in the order of their importance were as follows:

1.-Causes of Death

a) Post-of	erative hy	perthy	roic	lism	ar	d	sec	one	da	ry	C	ar	dia	ac	f	ail	ur	e	V	ve	re		
A. C. C.	ible for																						
) Primar	cardiac	failure	cau	sed																			4
) Pneum	nia cause	d								0.0												0 0	
) Emboli	sm cause	d																					
) Tetany	caused														0 0	* 0			0 #				
) Respira	ory failur	re cau	sed															0 1					
) Accide	t of intra	venous	inj	ectio	n c	au	sed											0 0		0 0	0 0		
) Wound	infection	and s	ecor	dary	pi	neu	mo	nia	١			0 0			0 0								
n) wound	mection	and 8	ecor	udry	pi	ieu	шо	1111			0 0	0 0	9 0		0 0	0 0		0 0	0 1	0 0	0 0		
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2.—Iodine, when administered as a pre-operative measure only, is a valuable factor in reducing mortality following thyroid operations. Iodine when administered in other than as a pre-operative measure, increases the danger of operation and renders the further intensive pre-operative treatment relatively ineffectual. Furthermore, indiscriminate treatment with iodine may obscure the fundamental seriousness of an underlying hyperthyroidism and may thus fail to warn the surgeon of a possible post-operative hyperthyroid crisis. Singularly, iodine thus administered may also cause acute exacerbation of symptoms, a condition which is most difficult to control and which yields poorly to the well-known measures of rest, sedatives, further intensive use of iodine and stage operations.

3.—Principal danger signals which should warn the surgeon are unusually acute hyperthyroidism or a prolonged period of hyperthyroidism, which has been present for years and which has reduced the vitality of the patient to a very low level; extreme cardiac damage with fibrillation and decompensation; the history and findings of chronic valvular disease; unfavorable history or findings referable to the respiratory system and such special factors as prolonged tracheal compression and recurrent nerve palsy.

4.—Incidental bad prognostic signs are extreme asthenia and loss of weight of twenty to fifty pounds or more; vomiting and diarrhea which indicate a severe intoxication with probable acidosis; unusually high basal metabolic rates; unusual nervous and mental symptoms such as great excitability, irrationalism and ideas of persecution, weakness and debility.

5.—Factors safeguarding the mortality are: refusal to operate in the presence of even the mildest respiratory infection; proper preparation of the

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patient; adapting the type and extent of operation to the tolerance of the patient; a carefully conducted operation with the idea of brevity consistent with safety; a proper regard for a rapid pulse, which tends to steadily increase during the course of the operation; avoidance of trauma to the recurrent nerves, parathyroid glands and trachea; complete control of hæmorrhage (I may digress to say that the control of hæmorrhage is of prime importance. We have had no death from hæmorrhage and in only one case was it deemed advisable to give a post-operative transfusion); further factors of safety are a well-conducted gas or local anæsthesia and a detailed post-operative care.

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FROM THE SECTION ON PATHOLOGIC ANATOMY OF THE MAYO CLINIC

ALTHOUGH statements concerning the vermiform appendix are supposed to have been made in ancient times, either from writings or inscriptions on tombs and monuments, or in the form of votive offerings, definite incontrovertible evidence of the existence of such knowledge was not discovered until the time of the Renaissance.

It seemed reasonable to believe that the presence of the appendix was well known when the pyramids were built, because all the viscera were removed from the body during the process of mummification and placed in four separate Coptic jars. In fact, certain Coptic jars in which the intestines were placed contained inscriptions on the exterior referring to the "worm" of the bowel. Herodotus, in the fifth century before Christ, during his visit to Egypt, stated that there were many "specialized physicians" among whom were those who specialized in diseases of the intestines. The "Hermetic books of Thoth," as well as the "Books of the dead," contained statements that in all probability referred to the appendix.

Greek votive offerings at Cos and Cnidus have been found which represented coils of intestines on which crude effort had been made to represent the appendix. Aristotle and Galen did not discover the appendix in their numerous physiologic researches, because they dissected only bodies of the lower animals. It should be remembered that an appendix comparable to that found in human beings occurs first in anthropoid apes, and it is fairly certain that they did not dissect such rare animals. Celsus, who was permitted by Tiberius Cæsar to dissect "executed" criminals, must certainly have discovered the presence of the appendix. Aretæus, of Cappadocia, in 30 A.D., is reputed to have described accurately an abscess of the appendix in which the patient recovered after simple incision and drainage of the abdominal wall.

In Italy, it was not until 1400 A.D. that dissection of the human body was publicly authorized, whereas it is stated on good authority that private dissections of the human body occurred in Germany during the latter half of the twelfth century.

FIRST Period.—Berengario da Carpi, in 1524, gave the first written account of the appendix which has been preserved. In 1543, the Fleming, Andreas Vesalius, professor of anatomy at Padua, accurately described and illustrated the normal appendix, with its relationship to other organs, in the magnificent "De fabrica humani corporis." When Vesalius left Padua and destroyed his books of anatomic drawings in a fit of anger, it was largely due to the efforts of his favorite pupil, Gabriele Falloppio, that physicians

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today possess many of the original teachings and anatomic descriptions made by Vesalius. Falloppio redrew and redescribed many of the original anatomic subjects which his teacher had destroyed. Vesalius called the appendix (more correctly from the Latin) "cæcum," meaning a blind pouch. This term persisted for a considerable period. Farnelius, in 1567, described a case of ileus resulting from a perforated appendix. Ambroïse Paré, the great French military surgeon, in 1582, following Vesalius, called the appendix the "cæcum." Bauhin, in 1597, in describing the ileocæcal valve, confused the appendix with the then unknown Meckel's diverticulum. Laurentine, in 1600, compared the appendix to a twisted worm. Vidius, in the same year, was first to describe the two muscular coats of the gastro-intestinal tract and its supporting mesentery. Fabricius, also in the same year, wrote an excellent account of the comparative anatomy of the ileocæcal region.

After this, information of further value was not added until 1706, when Morgagni, in the "Adversaria anatomia," devoted a large portion to the appendix, its normal site in adults, and its relations. However, he advanced the belief that the appendix was not of significance and had no function to perform. Verheye, in 1710, coined the term "appendix vermiformis" as a marginal heading in one of his writings. Santorini, in 1742, described the various positions of the appendix in the adult and illustrated several fecal concretions and worms found in his specimens. He concluded that the appendix served as a "nest" for worms of the gastro-intestinal tract. Lieberkühn, in 1739, published a classic essay on the appendix, in which he described for the first time the mucosal crypts, which today bear his name. Vosse, in 1749, described the various cæcal fossæ, and Weibricht described the valve situated at the juncture of the appendix and cæcum. This terminated the first period in the development of knowledge concerning the appendix, and it was characterized largely by anatomic studies. As yet, apparently nothing of its pathologic or physiologic significance was known.

Second Period.—In 1753, Heister first demonstrated, at necropsy, lesions occurring in the appendix. The first modern operation for an abscess of the appendix was done by a French surgeon, Mestivier, in 1759. At the subsequent necropsy it was found that a pin had perforated the wall and caused the abscess; fatal generalized peritonitis resulted. In the next fifty years, four necropsies were reported in the French literature; in all cases foreign bodies were found in the lumen of the appendix. Mestivier had originally pointed out that pathologic processes in the appendix would cause death. Haller, in 1778, stated that the fetal appendix was often as large in diameter as the ileum. In 1781, Sabatier drew attention to the fact that there was a large number of mucous glands in the appendix. At the beginning of the nineteenth century an English physician named Parkinson stated that perforation in the appendix should be considered as a frequent cause of death. Wegeler, in the same period, compared the fecal concretions found in the lumen of the appendix to gall-stones. In 1824, Louyer-Villermay published the observations found at necropsy in a clinical case of appendicitis.

In 1827, Melier, a French physician, presented a classic description of appendicitis and stated that it could cause primarily the lesions found in disease in the right lower quadrant of the abdomen. However, Dupuytren, the leading French surgeon of his day, together with his pupils, Husson, Dance and Menière, disagreed with Melier's statements and held him up to marked ridicule before French physicians. Melier, being a comparative youth, retracted his statements, and, as a consequence, medical advancement in regard to appendicitis was retarded for another period of approximately fifty years.

About 1830, Goldbeck and his German school advanced the belief that the primary disease in the ileocæcal region lay in the cæcum and not in the appendix. Thus, they advanced the terms "perityphlitis," "epityphlitis," and "endotyphlitis," in the hope that they were clarifying the situation. In reality, their efforts led to confusion and to a further retardation of the accurate clinical conceptions that appendiceal disease played. In 1837, Richards published the clinical data and the necropsy observations found in a typical case of acute perforated appendicitis in which the symptoms lasted for fifteen

days. In the same year, Hallowell described a similar case, with observations at necropsy, in which the appendicitis was a complication of pulmonary tuberculosis. Bright and Addison, about 1840, published excellent clinical descriptions of appendicitis. In 1846, Volz, another German, showed conclusively in forty post-mortem examinations of cases of appendicitis that the inflammation of the excum was always secondary to that of the appendix. However, his work was not widely accepted by his fellow countrymen at that time. This terminated the second period in the history of the appendix, characterized by pathologic studies which were as yet fundamentally incorrect in their conclusions.

Third Period.—In 1848, Hancock, of London, operated on a patient with acute appendicitis before the formation of abscess; a fecolith was found obstructing the lumen. Gay, an English physician, in 1850, was the first to expose to view a diseased appendix during an operation to relieve intestinal obstruction. Gerlach, in 1847, and in the years immediately subsequent, described the valve-like structure at the cæcal end of the appendix, which today bears his name. He gave one of the first excellent anatomic descriptions of the appendix. Lewis, of New York, in 1856, carefully reviewed the literature on abscesses of the appendix and foreign bodies found in the lumina. He failed, however, to appreciate their significance, and the relationship they might bear to appendicitis. Parker, in 1867, was credited with draining successfully four abscesses of the appendix. Bull, Matterstock and With, about 1880, recorded similar cases.

In 1886, Hall was the first American surgeon to remove a gangrenous appendix found accidentally in a strangulated inguinal hernia. In the same year, Fitz published the first 100 cases of successful drainage of abscesses of the appendix that covered the period from 1848 to 1886 that he was able to find in the literature of this country. From this work he showed, for the first time in a clear and decisive manner, that all inflammatory processes in the right lower quadrant of the abdomen should be considered, until proved otherwise, as originating in the appendix. He coined the term "appendicitis." Fitz's monograph is a classic and marked a milestone in the advance of surgery. With the publication of Fitz's paper, the third period in the history of the appendix was terminated. This period was characterized by simple drainage of abscesses of the appendix, which finally was done with increasing success by the more courageous surgeons. Pathologic studies finally had shown that appendicitis was significant and sensible rules were laid down for its successful treatment.

FOURTH PERIOD.—Woodbury stated that Morton, in 1887, was the first American to operate deliberately and successfully for appendicitis in a quiescent period. Sands, in 1889, diagnosed a ruptured appendix, operated, and sutured the site of the rupture. In 1889, McBurney published an excellent description of the pathologic changes in appendicitis, and described the abdominal area of maximal tenderness. He urged early operative interference. Lockwood and Rolleston, in 1891, described the various folds of the peritoneum and their relation to the position of the appendix. In 1892, Clado, a Frenchman, described a ligament which extends from the appendix as a peritoneal fold to the region of the right ovary, which it was stated affords direct lymphatic connection between the appendix and the right ovarian region, thus explaining how inflammations of the ovary or the appendix may involve the other structure by direct lymphatic extension. In 1892, Worcester wrote an excellent article on the rules for the treatment of appendicitis.

In 1893, Ribbert, of Germany, was the first to advance the belief that the appendix normally obliterated its lumen physiologically from the tip proximally toward the base. In 1894, Fowler is said to be the first American to publish a book on appendicitis; he reported 200 successfully performed operations. He stated that appendicitis is the most common cause of disease in the right lower quadrant in men. He likewise discarded the old terminology and pathologic descriptions that had been handed down for several centuries. Morris, in 1898, stated that the appendix was never a useful organ and that it was a threat to life when infected.

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In 1900, Montais, of France, contributed a study of chronic appendicitis, as did his fellow countryman, Rastouil, in the following year. In 1901, Berry, of England, showed by comparative anatomy that the appendix is not a vestigial structure, and Lockwood and Rolleston, also of England, wrote an excellent paper on the pathologic changes and treatment of chronic appendicitis. Fowler described his method of successful treatment for acute appendicitis, particularly when complicated by general peritonitis. In 1903, Faber, a German physician, described the mechanism of obliteration of the lumen of the appendix. Mertens described false diverticula of the sigmoid and appendix and mentioned the part they may play in acute inflammation. Aschoff, in the same year, described muscular defects in the wall of the appendix and showed the relation these defects might have to the formation later of diverticulum or as sites for rapid perforation in acute inflammation:

In 1903, Siredey published a study of the different forms of defects attributable to chronic appendicitis, and Faber stated that in cases of obliterated appendicitis, the farther away one is from the transitory zone in which obliteration occurred, the less evidence of inflammation one finds. Mundt published an article on the pathologic anatomy of the appendix, and Cameron described the various types of cystic dilatation and strictures that could occur following inflammatory processes of the lumen of the appendix.

In 1904, John B. Murphy advanced the well-known dictum, in cases of acute appendicitis, of opening the abdomen as quickly as possible and closing it more quickly. He advocated the administration of fluids by rectum as a means of giving rest to the gastro-intestinal tract in cases of generalized peritonitis. In the same year A. J. Ochsner published the details of his treatment of generalized peritonitis that resulted from appendicitis in an effort to induce localization of the peritonitis to the pelvis; this consisted of starvation, proctoclysis by the Murphy drip method, and administration of morphine. In 1904, von Brunn published his studies on the etiology of appendicitis and Hedinger wrote concerning congenital diverticula of the appendix. This fourth period of the history of the appendix was characterized by the firm establishment of logical, fairly successful methods of surgical treatment for acute appendicitis. The custom arose of removing the appendix under prophylactic conditions to prevent a possible attack of appendicitis in the future. The teachings of Fitz, Morton, McBurney, Ochsner, Murphy and Deaver, in regard to acute appendicitis, had been well learned by every surgeon. The same principles were sometimes incorrectly applied in the case of chronic appendicitis.

FIFTH PERIOD.—In 1905, Kelly and Hurdon published their excellent book on the vermiform appendix. The following year, Hansen advanced the belief that adhesions of the appendix to the adjacent structures, as well as the accumulation of fat in the meso-appendix, were evidences of chronic appendicitis. Waltner, of France, in 1908, stated that one form of chronic appendicitis could cause pseudo-anginal pain. In the same year Maale published his thesis on histopathologic studies of the appendix. His work, with its excellent photographs and drawings, is classic, and even today is of considerable value in the correct evaluation of pathologic changes found in the appendix.

In 1910, MacCarty presented a pathologic classification of appendicitis which showed that obliteration of the lumen occurs as the result of inflammation. Since, in many cases, the appendix becomes acutely inflamed during the process of obliteration, MacCarty urged that it would be safer to remove any appendix whose lumen was found to be obliterated. He likewise reported the incidence of carcinoma occurring in the appendix, in 22 of 5,000 specimens. This work was one of the first serious and extensive pathologic studies published in regard to appendicitis, particularly in its more chronic form. In the same year, Corner wrote concerning the function of the appendix, as well as the origin of appendicitis. Mummery, in 1910, stated that chronic appendicitis could be an etiologic factor in the causation of colitis, and Paterson and Moynihan agreed that chronic appendicitis often simulated gastric ulcer symptomatically. Austin

stated that duodenal ulcer was often accompanied by chronic appendicitis. Meyer believed that gastric disease could cause secondary pathologic changes in the appendix, and vice versa. Frilet, of France, pointed out the co-existence of cholecystitis with appendicitis. In 1911, MacCarty and McGrath wrote on the clinical and pathologic significance of obliteration of the appendix, based on the study of 5,000 cases. They showed that obliteration of the lumen of the appendix could occur much earlier than Ribbert had led investigators to believe. MacCarty and McGrath concluded that if persons reach the age of sixty years, the lumen of the appendix is either totally or partially obliterated. They stated that obliteration did not necessarily proceed proximally from the tip. It was their opinion that obliteration could be a pre-cancerous condition, because in their series of specimens carcinoma of the appendix occurred in cases in which the lumen was obliterated. Paterson stated that in 66 per cent. of ulcers of the duodenum the appendix was diseased and that it was without symptoms. André pointed out the relationship between diseases of the appendix and perforated duodenal ulcer, and Weber showed the relationship of appendicitis to the genesis of lesions of the gall-bladder and stomach.

In 1912, Moynihan stated that most peptic ulcers originate from a previously diseased appendix. Singer, in the same year, described the effect of chronic appendicitis on the secretory activity of the stomach, and its etiologic rôle in the causation of gastric disease. In 1913, Roux wrote a thesis on the employment of Röntgen-rays as a method of diagnosis for appendicitis. He was able to visualize a high percentage of foreign bodies and fecal concretions in the lumen. In the same year, Paterson stated that in cases of chronic appendicitis hæmatemesis and hyperchlorhydria ceased after appendectomy. Lockwood concluded that achylia resulted from chronic appendicitis in 6 per cent. of cases, whereas in an additional 12 per cent. more than 300 cubic centimetres of gastric secretions were contained in a stomach at rest prior to appendectomy. Illoway demonstrated the relationship between hyperacidity and chronic appendicitis.

Thus it is apparent that up to 1913, the pendulum had swung far to an extreme, and the appendix was blamed as the cause of numerous pathologic conditions which later investigation minimized. We are indebted, in this early period, to the pioneer work done by Fitz, Price, Morton, Fowler, Morris, Murphy, Ochsner, Richardson and Brewster, and Deaver, in establishing the proper regard for the early treatment of appendicitis. However, appendectomy was frequently being performed, without sufficient indication, by the general practitioner. This fifth period of history of the appendix was characterized by perfection of surgical technic. However, the diagnosis of chronic appendicitis and its surgical treatment had become overemphasized.

Sixth Period.—In 1914, Stanton reported that, in a series of appendectomies performed by him, in 36 per cent. of cases in which the condition was chronic, relief was not experienced following operation. Pringle stated, in the same year, that many of the bands found around the ileocæcal region which caused chronic intestinal stasis were embryonic and not inflammatory. W. J. Mayo, in 1914, stated that the appendix is a lymphoid organ and that it undergoes normal regression at the end of puberty. Thus, carcinoma of the appendix, occurring during childhood or adolescence, is more malignant and metastasizes more quickly than that which occurs in the appendix of the adult because of the marked lymphoid hyperplasia. This had been previously pointed out by C. H. Mayo in 1903. Adami stated that Bacillus coli and streptococci used experimentally could produce fibrosis, and it was his belief that these two organisms are more or less responsible for the fibrosis found in cases of chronic appendicitis. Seedham and Green stated that chronic appendicitis often causes symptoms typical of peptic ulcer. Mathieu stated that chronic appendicitis is often associated with chronic colitis.

In 1915, Rosenow reported that streptococci isolated in the appendix exhibited immediate elective localization in 68 per cent. of rabbits that received injections, and showed the same result in an additional 15 per cent. at a later period. MacLaren distinguished gynæcologic conditions and virginal ptosis from the syndrome assigned to

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chronic appendicitis. Deaver and Aaron stated that chronic appendicitis could easily simulate pylorospasm and duodenal ulcer symptomatically. Berceanu demonstrated that a direct lymphatic connection by way of the pancreatic region existed between the appendix and the first portion of the duodenum.

In 1916, Connell stated that the diagnosis of chronic appendicitis was being made too recklessly and without sufficient observation. In 1918, Evans stated that in 91 per cent. of 236 cases of acute appendicitis there was no history of previous attack, whereas in 86 per cent. there was a history of recent acute infection of the upper respiratory tract. Detweiler and Maitland stated that streptococci isolated in cases of appendicitis showed elective localization in 14 per cent. of animals that received injections. Brown, in 1919, stated that chronic appendicitis is the result of visceroptosis, poor circulation, stasis and infection.

In 1919, Williams and Slater, in a series of 500 consecutive laparotomies, found that adhesions, fibrosis or kinks in the appendix were present in 33 per cent. of all patients undergoing gynæcologic operations. Billings, in 1920, stated that appendicitis could be caused by an infection of the blood-stream. Hall and Dyas, at Camp Logan, Texas, during the epidemic of influenza in 1918, found that the incidence of appendicitis rose to 440 per cent. above the usual incidence. Stanton, in 1919, in a study of results after appendectomy in cases of chronic appendicitis, found that the end-results were poor, so far as relief of symptoms was concerned. Carman, in 1920, stated that since in few cases the appendix is normal, the diagnosis of chronic appendicitis is accurate with moderate röntgenologic evidence.

Cabot, in 1920, stated that the "chronic appendix" had become the scapegoat of abdominal surgery. Pitzman stated that an appendix should not be removed unless an organic stricture could be demonstrated. In the same year, Deaver reported that chronic appendicitis could cause pylorospasm, hyperacidity and indigestion. In 1921, Vasselle and Parturier stated that chronic appendicitis could be the cause of hæmatemesis and melena. Masson, also in 1921, stated as his belief that the symptoms of pain in an appendix with an obliterated lumen are due to the formation of neuromas arising from the plexuses of the sympathetic nervous system or from cells staining with silver salts in the depths of the crypts of Lieberkühn.

O'Neil, in 1921, showed that in 25 per cent. of cases of ureteral calculi in the Massachusetts General Hospital, operation was first performed for chronic appendicitis. Rosenow, in the same year, again showed that streptococci, isolated from cases of chronic appendicitis, showed elective localization in 70 per cent. of animals that received injection. In 1921, Battle, in a study of 1,000 cases in which appendectomy was performed during the quiescent interval, found that the appendix in 10.8 per cent. was normal on microscopic examination. Nicholson, in the same year, made a plea for the normal appendix and reviewed its physiologic rôle in the metabolism of the body. Moynihan, in reviewing his cases of chronic appendicitis, found a high percentage of changes in the appendix, especially in those accompanied by peptic ulcer. Sanes, in 1922, reported that in three cases of ureteral calculi which came under his observation, appendectomy was first performed. Chalfant stated that the ultimate results of appendectomy in chronic cases were the poorest in all surgical practice. Masson felt that the lesions of the appendix resembling neuromas found in the plexuses of periglandular nerves were responsible for the symptoms of chronic appendicitis. McCarrison felt that appendicitis was due to the refined food ingested by civilized peoples. Balfour reported cases of hæmatemesis that were apparently associated with appendicitis. In the same year, Whiteford stated that in the better interests of surgery and the patient operation for a chronically inflamed appendix should be abandoned. Lichty reported an occurrence of 40 per cent. failures of symptomatic cures in cases in which the diagnosis before operation was chronic appendicitis. Nicolle and Conseil, of France, showed that individual immunity against bacillary dysentery and Malta fever is partially carried in the lymphoid tissue of the appendix.

In 1923, Digby stated that the appendix had an immunizing function against bacterial poisons in the cæcum. Ehrlich felt that a diagnosis of chronic appendicitis was justified if the ascending colon did not empty six hours after a barium meal, or after another meal had been taken by mouth. Deaver and Raydin reported a series of 500 cases of chronic appendicitis in which treatment was by surgical methods; microscopic examinations showed that 3.8 per cent. of the specimens were normal. The mortality was 0.27 per cent. for this series. Braithwaite, in the same year, by injection experiments, showed that there is a direct lymphatic path from the appendix along the superior mesenteric vessels extending to the lymph-nodes around the pyloric region of the stomach. Doolin, in the same year, reported that operation for chronic appendicitis in 33 per cent. of his cases was a failure so far as relief of symptoms was concerned. Melchior and Loser stated that in only 60 per cent. of their cases in which operation was performed was improvement noted. Herrick stated that 40 per cent, of patients seen by him possessed cutaneous hypersensitiveness in the right lower quadrant of the abdomen from pyelitis, ureteritis or hydronephrosis, and the condition was commonly mistaken for chronic appendicitis.

In 1924, W. J. Mayo stated as his belief that the appendix bore a definite relationship to, and often was the etiologic agent of, other abdominal lesions. Heyd concurred in this opinion, and expressed the belief that the symptoms of appendicitis commonly mimic peptic ulcer, and also cause definite resultant pathologic changes in the liver. In the same year C. H. Mayo advised against appendectomy as a routine in extensive pelvic operations when there was considerable peritoneal effusion, for fear of causing generalized peritonitis. Paus subscribed to this, and stated that chronic appendicitis, in addition, commonly simulated adnexitis and cholecystitis. Davison and Royer expressed the belief that the symptoms of adhesions surrounding the ascending colon commonly simulate those of chronic appendicitis. Brulé, of France, concluded that diseases of the colon also simulated chronic appendicitis, that diet and medical treatment were the only therapeutic agents of value, and that appendectomy was contra-indicated. Bettmann listed numerous diagnostic errors leading to unwarranted operations of appendectomy. In a series of 170 cases observed post-operatively he found that in 33 per cent. in which the diagnosis before operation was chronic appendicitis, relief of symptoms was not obtained. Connell felt that numerous conditions, such as visceral ptosis, constipation and neurasthenia were commonly confused with chronic appendicitis, and needless operations were performed that did not afford relief. Connell and Gibson reported that in only 61 per cent. of their cases in which operation had been performed following a diagnosis of chronic appendicitis improvement did not follow surgical intervention.

In 1925, Sherren, of England, stated that symptoms often caused by virginal ptosis and chronic peptic ulcer lead to a diagnosis of chronic appendicitis. Schoemaker stated that vascular congestion of the pylorus, in forty-five of his cases, was responsible for symptoms of pseudo-appendicitis, and that of sixteen appendixes removed only four gave microscopic evidence of pathologic changes. All of his patients operated on for chronic appendicitis were not relieved of symptoms. Horsley, in the same year, concluded that chronic ulcer of the terminal portion of the ileum commonly simulated chronic appendicitis symptomatically. Shutt expressed the belief that adhesions of the colon were a factor in the failure to cure the symptoms in cases in which appendectomy was performed for chronic appendicitis. He felt that unless these adhesions were removed, the patient's symptoms would probably not improve. Schnitzler was of the opinion that the so-called chronic appendix did not exist. Deaver, on the other hand, believed chronic appendicitis to be a clinical entity and divided it into two distinct types: That which occurred after acute appendicitis, and that which has always taken the form of chronic inflammation without acute exacerbations. He felt that there could be a differential diagnosis in these two types of chronic appendicitis. He stated his belief that in cases of chronic peptic ulcer, chronic cholecystitis, ureteral calculi, Dietl's crisis and chronic pelvic inflammatory disease, that the appendix is commonly unjustifiably removed.

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In 1926, Hertzler felt that fibrosis in the so-called chronic appendix did not cause symptoms, and that the term "chronic appendicitis" was not founded on pathologic facts. He thought that the alleged relief that resulted after operation in such cases was not proof of the existence of such a clinical entity. He expressed as his belief that most patients, if observed over a considerable period of time, did not obtain permanent cure, and it was his opinion that the treatment in such cases was in making an accurate clinical diagnosis of the true condition present, and that surgical intervention was not justifiable. Hathaway, of England, in the same year, said that the day of so-called chronic appendicitis was past, and that pain at McBurney's point did not mean the existence of chronic inflammation of the appendix. He expressed as his belief that the only true chronic appendicitis was that due either to tuberculosis or to actinomycosis. He also felt that perityphlitis, a dilated or mobile execum, or excal stasis frequently gave rise to symptoms simulating chronic appendicitis. Ramond and Parturier, of France, reported instances of chronic cholecystitis in cases in which the gall-bladder occupied the right lower quadrant of the abdomen, and in which symptoms and signs caused a false diagnosis of chronic appendicitis. Harrenstein, of Germany, believed that insufficiency of the ileocæcal valve was the essential factor in the entire problem of chronic appendicitis. W. J. Mayo, in 1926, felt that there were two types of chronic appendicitis: That in which the appendix contained fecal concretions, and that in which it contained fibrotic material with fibrous adhesions surrounding it. Neither type commonly caused pain, but he believed that surgical removal was indicated in both types. Morris classified chronic appendicitis into five types: Fibroid; that resulting from the lack of peritoneal support; that resulting from irritation of a contracted scar; that resulting from lymphoid hyperplasia of the lumen; and catarrhal, with an accompanying infection of the cæcum. In 1926, Codman reported that in only 61 per cent. of appendixes removed by him were microscopic pathologic changes noted. Coffey reported no improvement in 70 per cent. of his cases in which operation had been done elsewhere for chronic appendicitis.

In 1927, Butka, in a pathologic study of 202 appendixes removed at operation from patients with chronic appendicitis, found that pathologic change was absent in 66 per cent. Harrenstein expressed as his belief that adhesions around the appendix found at the time of operation were not of value in the diagnosis of chronic appendicitis. Payr, of Germany, felt that primary chronic appendicitis is usually cured symptomatically by a medical regimen of athletic exercises and massage in cases of enteroptosis, and that in such cases the condition is made worse by appendectomy. However, he believed that there are a few cases in which the diagnosis of chronic appendicitis is justified. Fraikin, of France, stated his belief that cæcal spasm is commonly responsible for the diagnosis of chronic appendicitis. Visani, of Italy, advanced the theory that certain toxic nervous factors in chronic appendicitis later lead to cholecystitis. Guénaux and Vasselle stated that only 66 per cent, of normal appendixes are röntgenographically visualized with the barium meal. Chase expressed the belief that diverticula of the appendix often lead to the diagnosis of chronic appendicitis. Walton, of England, thought that it was impossible for an appendix to remain chronically inflamed for years. He had performed 1,738 laparotomies in which the appendix was not removed, and of that number, in a follow-up period of ten years, acute appendicitis developed in only five cases that required appendectomy. Trotter, of England, stated that if the appendix is elongated, adherent to adjacent structures, of irregular calibre, or contains fecoliths, appendectomy should be performed. Lichty, in 1927, again drew attention to the fact that the appendix in middle life normally begins to be replaced by fibrous tissue, and that the end-result of such a process is a mere fibrous cord. Bonney is of the opinion that many gynæcologic conditions may simulate chronic appendicitis, and warns that this must be constantly kept in mind. Carnett, in the same year, again drew attention to the fact that in many cases in which the diagnosis was chronic appendicitis, the condition really was caused

by intercostal neuralgia, sacro-iliac strain and root pain of the spinal nerves of the lumbar region.

In 1928, Rogalsky, of Russia, published a study of involution in the appendix. Saykoff stated that the results of operation for chronic appendicitis in Russia were rather poor. Shapiro drew attention to the fact that children commonly have many ailments that simulate appendicitis and that appendectomy is the worst possible treatment. Aschoff, in 1928, said that chronic appendicitis, of itself, was not a separate disease, but was the end-result of repeated attacks of acute appendicitis. From his experience he believed that in 62 per cent. of cases the normal appendix contains either feces or fecoliths. W. J. Mayo, in 1928, felt that the appendix was a member of the reticulo-endothelial system which normally retrograded to a senescent type in middle age. He said that the appendix normally undergoes involution and that, therefore, "appendicitis obliterans" in middle-aged persons is a poor term for a normal physiologic process. Andresen, in the same year, reported a series of 230 patients on whom appendectomy had been performed previously because of gastro-intestinal symptoms, and a later, more complete study revealed that peptic ulcer was present in thirty-six, gastritis or duodenitis in twenty-six, cholecystitis in thirty-five, colitis in twenty-one, adhesions in twenty-five, hernia in seven, and carcinoma in seven. He advised an essentially medical treatment in most cases, with elimination of the foci of infection and proper dietetic and hygienic care.

In 1928, Collins drew attention to the rôle that non-malignant abnormalities and constricting bands of the ascending colon may play in simulating vague symptoms falsely diagnosed as due to peptic ulcer, cholecystitis, or appendicitis. It was his plea that if these organs were found to be more or less of normal appearance on exploratory laparotomy, that the ascending colon be carefully examined for the presence of pathologic abnormalities responsible for the patient's symptoms. In the same year, Deaver stated that adhesions around the appendix are common evidence of chronic appendicitis, especially those involving the terminal portion of the ileum, and causing Lane's kink. He said the lumen was first obliterated by hyperplasia of the submucosa that resulted from mural inflammation followed by exudation, organization, and finally replacement of connective tissue, or appendicitis obliterans. Carnett and Boles wrote concerning the fallacy of diagnosing chronic appendicitis. They believed this to be a disease of the entire gastro-intestinal tract, and that it is not cured merely by appendectomy. They stated that since röntgenologists do not agree as to what constitutes a positive diagnosis of chronic appendicitis by fluoroscopy, that their help is of questionable value. They likewise drew attention to the huge economic loss suffered by this country each year because of needless appendectomies. Menon, of India, stated that in chronic appendicitis there is atrophy of lymphoid tissue, which normally does not occur in human beings before the age of fifty years. He noted that lymphocytes are normally found in considerable numbers in the submucosa. He felt that a definite relationship existed between lymphoid hyperplasia of the appendix and colic-like pain in the abdomen. Bettmann, in 1928, compiling figures from thirty-five leading hospitals in the central portion of the United States in 1927, found that there were 5,664 appendectomies done for chronic appendicitis; the symptoms in 40 per cent. were unrelieved, and in 11.6 per cent. the symptoms were worse following operation. The immediate operative mortality was 1.7 per cent.

In 1929, Deaver stated that a diagnosis of chronic appendicitis should be made only after careful evaluation of the patient's symptoms, and that all confusing factors should first be ruled out before surgical intervention is undertaken. Menon stated that the presence of eosinophils in the mucosa of the appendix is within normal limits, at least in the tropics. He stated, however, that numerous eosinophils in the submucosa constitute definite evidence of chronic appendicitis. Mallory mentioned that the condition of the appendix in various stages of repair is often spoken of as chronic appendicitis, but that the term is not justifiable. Reischauer, in 1929, suggested that the initial

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cause of appendicitis is a disturbance in the neurocirculatory system of the appendix, which, in turn, results in pathologic changes in tissue, favoring the growth of bacteria in the lumen. Freedman, in the same year, reported a series of forty-two cases of children in which acute appendicitis developed during an epidemic of acute infection of the upper respiratory tract. He suggested that there may be an etiologic relationship between these two diseases.

In 1930, Dorsey reported that streptococci isolated from appendixes removed at operation resemble morphologically and culturally streptococci isolated from the nasopharynx of patients with appendicitis, and streptococci obtained from the tonsils of patients with arthritis. Inoculation of animals with streptococci from patients with appendicitis gave a high degree of localization in the appendix, whereas localization in the joints was low. She believed that appendicitis may be a hæmatogenous, intramural, streptococcal infection, with the nasopharynx as the source of the infection. Ballenger reported acute appendicitis developing in two cases on the second day following removal of diseased tonsils. Rosenow again drew attention to the high degree (60 per cent.) of elective localization occurring in animals that had received injections of streptococci isolated from cases of chronic appendicitis. Alvarez directed attention to the part that mesenteric lymphadenitis in adults can play simulating chronic appendicitis symptomatically. Askanazy and Bamatter, in 1930, wrote a comprehensive report on the pathologic changes of chronic forms of appendicitis. Aschoff, in his recent book on the pathology of the appendix, devotes considerable space to the chronic types of appendicitis and the bacterial fauna found in such appendixes. He believes, as does Weinberg, of France, that possibly anaërobic bacteria may play a considerable part in the toxic symptoms referable to chronic appendicitis as well as to the occasional profound toxemia encountered in acute cases. In 1931, Cooke wrote a complete article on carcinoid tumors of the appendix, reviewed from the literature and from cases seen in The Mayo Clinic.

Seventh Period.—The seventh period of the history of the appendix has now arrived. The treatment of acute appendicitis has been on a sound basis for the last thirty years, and little in the way of improvement has been added during the last twenty. Humanity owes an incalculable debt of gratitude to the pioneer work of men such as Fitz, Morton, Fowler, Ochsner, Murphy, Morris, Price, Richardson, Moynihan and W. J. Mayo. The name of one man, during the last forty years, stands preëminently above all others in the field of appendicitis. The medical world owes a debt to Deaver, who has placed the treatment of appendicitis on such a firm foundation that it is now common knowledge, even to students who are beginning their medical careers.

The battle over the treatment of chronic appendicitis has continued for the last twenty years. It has excited keen controversy, but during the last three years the conviction has been gaining ground that the diagnosis of chronic appendicitis must be made only after the history has been very carefully taken and thorough exclusion has been made of the numerous conditions simulating appendicitis. After the diagnosis has been made, and operation decided on, it must be remembered that in a certain percentage of cases symptomatic relief will not be complete after operation, and that the pathologic study of the removed appendix may show little, if any, change from normal. The day of indiscriminate appendectomy has passed, as have many of the former empiric procedures which failed to stand the impartial scrutiny of modern scientific investigation into their end-results.

BIBLIOGRAPHY

- Aaron, C. D.: Chronic Appendicitis, Pylorospasm and Duodenal Ulcer; A Preliminary Note. J. A. M. A., vol. lxiv, p. 1845, May 29, 1915.
- ² Adami, J. G.: An Address on Chronic Intestinal Stasis; "Auto-intoxication" and Sub-infection. Brit. Med. Jour., vol. i, pp. 177–183, January 24, 1914.
- ⁸ Alvarez, W. C.: Mesenteric Lymphadenitis in Adults, a Cause of Pseudo-appendicitis, Indigestion, Diarrhœa, and Arthritis. Med. Clin. N. Amer., vol. xiv, pp. 605–617, November, 1930.
- ⁶ André: Appendicite; ulcère perforé du duodénum; abcès sous-phrénique. Clinique Brux, vol. xxv, p. 253, 1911.
- ⁶ Andresen, A. F. R.: Medical Aspects of Chronic Appendicitis. Am. Jour. Surg., vol. v, pp. 372–377, October, 1928.
- ⁶ Aschoff, Ludwig: Muskelwanddefekte am Processus vermiformis. Deutsch. med. Wchnschr., vol. xxviii, p. 250, August, 1902.
- ⁷ Aschoff, Ludwig: Über chronische Appendicitis. Med. Klin., vol. xxiv, pp. 1660-1661, October 26, 1928.
- ⁸ Aschoff, Ludwig: Der appendicitische Anfall; seine Atiologie und Pathogenese. Julius Springer, Berlin, 125 pp., 1930.
- ⁹ Askanazy, M., and Bamatter, F.: Einige Formen chronischer Wurmfortsatzentzündung. Virchow's Arch. f. path. Anat. u. Physiol., vol. cclxxv, pp. 652–673, 1930.
- ²⁰ Austin, H. W.: Duodenal Ulcer with Perforation Accompanied by Chronic Appendicitis; Operation Twelve Hours After Perforation of Duodenum for Removal of Appendix and Closure of Perforation; Recovery. Med. Rec., vol. 1xxvii, p. 708, April 23, 1910.
- ¹¹ Balfour, D. C.: Hematemesis. Tr. Coll. Phys., Philadelphia, s. 3, vol. xliv, pp. 236-254, 1922.
- ¹² Ballenger, H. C.: Appendicitis Following Tonsillectomy. Report of Two Cases. Arch. Otolaryngol., vol. xii, pp. 67–71, July, 1930.
- ¹³ Battle, W. H.: Operation for Removal of the Appendix in the Quiet Period. Lancet, vol. i, pp. 313-316, February 12, 1921.
- ¹⁴ Berceanu, Dan: Les relations lymphatiques entre l'appendice et la région duodénopancréatique. Rev. de chir., vol. 1xii, pp. 356-361, 1924.
- ¹⁵ Berry, R. J. A.: The True Cæcal Apex: An Attempt to Prove that the Vermiform Appendix Is Not a Vestigial Structure. Brit. Med. Jour., vol. i, p. 648, March 16, 1901.
- Bettmann, H. W.: Diagnostic Errors Leading to Uncalled-for Appendectomy. J. A. M. A., vol. lxxxiii, pp. 1216-1221, October 18, 1924.
- ¹⁷ Bettmann, H. W.: Chronic Appendicitis from the Viewpoint of an Internist. Ann. Int. Med., vol. ii, pp. 509-517, December, 1928.
- ¹⁸ Billings, Frank: Focal Infection. In: Oxford Medicine, vol. i, pt. 1, pp. 155–176, New York, Oxford University Press, 1920.
- ¹⁹ Bonney, Victor: Discussion on Chronic Appendicitis: Gynæcologic Considerations in Chronic Appendicitis. Brit. Med. Jour., vol. ii, pp. 1066–1067, December 10, 1927.
- ²⁰ Braithwaite, L. R.: The Flow of Lymph from Ileocæcal Angle, and Its Possible Bearing on the Cause of Duodenal and Gastric Ulcer. Brit. Jour. Surg., vol. xi, pp. 7-26, July, 1923.
- ²¹ Bright, Richard, and Addison, Thomas: Elements of the Practice of Medicine. Longman, vol. i, 498 pp., London, 1839.
- ²² Brown, T. R.: Late Results of Supposedly Successful Abdominal Operations on the Digestive Tract. J. A. M. A., vol. lxxiii, pp. 1501–1506, November 15, 1919.
- ²⁸ Brulé, M.: L'appendicite chronique. Presse méd., vol. xxxii, pp. 893–895, November, 1924.

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²⁶ von Brunn, Max: Beiträge zur Aetiologie der Appendicitis. 1. Bedeutung der Veranderungen der Blut— und Lymphgefässe des Mesenteriolum. 2. Bedeutung der Kotsteine. Beitr. z. klin. Chir., vol. xlii, pp. 61–186, April, 1904.

Bull, W. T.: Perityphlitis. New York Med. Jour., vol. xviii, pp. 240-264, Sep-

tember, 1873.

²⁵⁰ Butka, H. E.: Chronic Appendicitis: A Study of 202 Consecutive Cases. California and West. Med., vol. xxvi, pp. 467–470, April, 1927.

²⁸ Cabot, Hugh: Chronic Appendicitis, the Scape-goat of Abdominal Surgery. Jour. Michigan State Med. Soc., vol. xix, pp. 452-456, October, 1920.

²⁷ Cameron, H. C.: Appendix Showing Cystic Dilatation and Stricture. Glasgow Med. Jour., vol. 1x, pp. 423–425, 1903.

²⁸ Carman, R. D.: The Röntgen Diagnosis of Diseases of the Alimentary Canal. W. B. Saunders Company, Ed. 2, 676 pp., Philadelphia, 1920.

²⁹ Carnett, J. B.: Chronic Pseudo-appendicitis Due to Intercostal Neuralgia. Am. Jour. Med. Sc., vol. clxxiv, pp. 579–599, November, 1927.

³⁰ Carnett, J. B., and Boles, R. S.: Fallacies Concerning Chronic Appendicitis. Tr. Sect. Gastroenterol. and Proctol., Am. Med. Assn., pp. 150-162, 1928.

⁸¹ Chalfant, S. A.: Some Gynæcologic Misdemeanors. J. A. M. A., vol. 1xxviii, pp. 1675–1676, June 3, 1922.

⁸⁸ Chase, W. H.: Three Cases of Diverticula of the Appendix. Canad. Med. Assn. Jour., vol. xvii, pp. 416-420, April, 1927.

83 Clado: Appendice caecal. Anatomie, embryologie, anatomie comparée, bactériologie normale et pathologique. Compt.-rend. Soc. de biol., vol. xliv, pp. 133–172, 1892.

⁵⁴ Codman, E. A.: Quoted by Vaughan, R. T.: Appendicitis. In: Nelson's Loose-leaf Living Surgery. Thomas Nelson and Sons, vol. v, pp. 297-342, New York, 1928.

Coffey, R. C.: Gastroenteroptosis. D. Appleton and Co., 153 pp., New York, 1923.
 Coffey, R. C.: Quoted by Vaughan, R. T.: Appendicitis. In: Nelson's Loose-leaf Living Surgery. Thomas Nelson and Sons, vol. v, pp. 297–342, New York, 1928.

See Collins, F. K.: The Ascending Colon. California and West. Med., vol. xxviii, pp. 179–183, February, 1928.

Connell, F. G.: Pseudo-appendicitis. J. A. M. A., vol. lxvii, pp. 335–338, July 29, 1916.
 Connell, F. G.: Chronic Appendicitis. Wisconsin Med. Jour., vol. xv, pp. 300–316,

February, 1917.

⁸⁰ Connell, F. G.: Chronic Appendicitis. President's Address. Wisconsin Med. Jour., vol. xxii, pp. 371-374, January, 1924.

⁴⁰ Connell, F. G.: Chronic Appendicitis. Journal-Lancet, vol. xlv, pp. 7-12, January 1, 1925.

⁴¹ Cooke, H. H.: Carcinoid Tumors of the Small Intestine. Arch. Surg., vol. xxii, pp. 568-597, April, 1931.

⁴² Corner, E. M.: The Function of the Appendix and the Origin of Appendicitis. Annals of Surgery, vol. lii, pp. 512-519, October, 1910.

⁴⁹ Davison, Charles, Davison, Marshall, and Royer, D. J.: Adhesions About the Ascending Colon Simulating Chronic Appendicitis. Surg., Gynec. and Obstet., vol. xxxviii, pp. 171–180, February, 1924.

⁴⁴ Deaver, J. B.: Remarks Upon Some Points in the Technic of the Operation for Appendicitis. Annals of Surgery, vol. xxvii, pp. 78-82, January, 1898.

⁴⁵ Deaver, J. B.: Appendicitis. P. Blakiston's Son and Company, Ed. 4, 379 pp., Philadelphia, 1913.

⁴⁶ Deaver, J. B.: Chronic Appendicitis Simulating Duodenal Ulcer. Internat. Jour. Surg., vol. xxix, pp. 377–382, December, 1916.

⁴⁷ Deaver, J. B.: Chronic Appendicitis. Med. Clin. N. Amer., vol. iii, pp. 1167–1174, March, 1920.

⁴⁰ Deaver, J. B.: Appendicitis Then and Now. Therap. Gaz., s. 3, vol. xxxvi, pp. 533-537, August, 1920.

- ⁴⁰ Deaver, J. B.: Chronic Appendicitis. Surg. Clin. N. Amer., vol. v, pp. 1514–1516, December, 1925.
- Deaver, J. B.: Chronic Appendicitis. Internat. Clin., s. 38, vol. ii, pp. 210-213, June, 1928.
- ⁶¹ Deaver, J. B.: Chronic Appendicitis. Am. Jour. Med. Sc., vol. clxxvii, pp. 749-758, June, 1929.
- E Deaver, J. B., and Ravdin, I. S.: End-results of Five Hundred Cases of Chronic Appendicitis: A Statistical Study. Arch. Surg., vol. vi, pp. 31-40, January, 1923.
- Detweiler, H. K., and Maitland, H. B.: The Localization of Streptococcus Viridans. Jour. Exper. Med., vol. xxvii, pp. 37-47, January, 1918.
- ⁵⁴ Digby, K. H.: Additional Notes on Immunizing Function of the Sub-epithelial Lymphatic Glands. Lancet, vol. ii, pp. 1077–1078, November 17, 1923.
- Doolin, William: Some Unsatisfactory Appendicectomies. Irish Jour. Med. Sc., s. 5, pp. 495-503, January, 1923.
- Dorsey, Anna H. E.: Bacteriology and Pathogenesis of Appendicitis. Surg., Gynec. and Obstet., vol. 1, pp. 562-571, March, 1930.
- ⁶⁷ Dupuytren, Guillaume: Leçons orales de clinique chirurgicale. Germer-Baillière, vol. iii, pp. 330-341, Paris, 1833.
- Ouputryen, G.: On Lesions of the Vascular System, Diseases of the Rectum, and Other Surgical Complaints, Being Selections from the Collected Edition of the Clinical Lectures. New Sydenham Society, pp. 294-310, London, 1854.
- Ehrlich, Franz: Röntgendiagnose der sogennanten Appendicitis chronica. Deutsch. med. Wchnschr., vol. xlix, p. 449, April 6, 1923.
- ⁶⁰ Evans, J. S.: Epidemiology of Acute Appendicitis in Relation to Acute Nasal and Tonsillar Infections. Wisconsin Med. Jour., vol. xvii, pp. 91-93, August, 1918.
- 61 Faber, Knud: Appendicitis Obliterans. Hospitalstidende, vol. x, pp. 867–878, 1902.
- Eaber, Knud: Über Appendicitis Obliterans. Mitt. a. d. Grenzgeb. d. Med. u. Chir., vol. xi, pp. 506-530, 1903.
- ⁶³ Fitz, R. H.: Perforating Inflammation of the Vermiform Appendix with Special Reference to Its Early Diagnosis and Treatment. Tr. Assn. Am. Phys., pp. 107– 144, 1886.
- ⁶⁴ Fitz, R. H.: The Relation of Perforating Inflammation of the Vermiform Appendix to Perityphlitic Abscess. New York Med. Jour., vol. xlvii, pp. 505-508, 1888.
- ⁶⁶ Fitz, R. H.: Appendicitis: Some of the Results of the Analysis of Seventy-two Cases Seen in the Past Four Years. Boston Med. and Surg. Jour., vol. exxii, pp. 619–620, June 19, 1890.
- 66 Fowler, G. R.: Observations Upon Appendicitis. Ann. Surg., vol. xix, pp. 4-46, 146-171, 327-362, 475-491, 546-585; 1894.
- ⁶⁷ Fraikin, A.: Les espines irritatives abdominales: le spasme du caecum dans l'appendicite chronique. Paris méd., vol. Ixiii, pp. 414-415, April, 1927.
- ⁶⁸ Freedman, H. J.: Forty-two Cases of Appendicitis in Children Occurring During an Epidemic of Upper Respiratory Tract Infection. Arch. Pediat., vol. xlvi, pp. 604– 616, October, 1929.
- ⁶⁰ Frilet, Eugène: De la coexistence de la cholécystite et de l'appendicite (cholécysto-appendicite). Lyon, 88 pp., 1910.
- Garrison, F. H.: An Introduction to the History of Medicine. W. B. Saunders Company, Ed. 3, 942 pp., Philadelphia, 1921.
- ⁷¹ Gay, John, Internal Strangulation Between the Appendix Vermiformis, Which Had Become Adherent to the Ileum, and a Band of False Membrane: Operation. Tr. Path. Soc. London, vol. iii, pp. 101–106, 1850–1852.
- Gerlach, J.: Tödliche Peritonitis, als Folge einer Perforation des Wurmfortsatzes. Ztschr. f. rat. Med., vol. vi, pp. 12-23, 1847.
- Gerlach, J.: Zur Anatomie und Entwickelungsgeschichte des Wurmfortsatzes. Wissensch. Mitt. d. phys.-med. Soc. zu Erlangen, vol. i, pp. 7-16, 1859.

- ⁷⁴ Gibson, G. L.: The Results of Operations for Chronic Appendicitis: A Study of 555 Cases. Am. Jour. Med. Sc., vol. clix, pp. 654-663, May, 1920.
- ⁷⁵ Gibson, G. L.: The Results of Operations for Chronic Appendicitis (Second Series). Am. Jour. Med. Sc., vol. claviii, pp. 807–812, December, 1924.
- ⁷⁶ Goldbeck, Gottfried: Ueber eigenthümliche entzündliche Geschwülste in der rechten Hüftbeingegend. J. A. Kranzbühler, 30 pp., Worms, 1830.
- ⁷⁷ Guénaux, G., and Vasselle, P.: Le radio-diagnostic de l'appendicite chronique. Paris méd., vol. lxiii, pp. 141-147, February, 1927.
- ⁷⁸ Hall, J. N., and Dyas, F. G.: Appendicitis at Camp Logan as a Sequel to Influenza and Pneumonia. J. A. M. A., vol. 1xxii, pp. 726-727, March 8, 1919.
- Hall, R. J.: Suppurative Peritonitis Due to Ulceration and Suppuration of the Vermiform Appendix; Laparotomy; Resection of the Vermiform Appendix; Toilette of the Peritonaeum; Drainage; Recovery. New York Med. Jour., vol. xliii, pp. 662-663, June 12, 1886.
- Hallowell, Edward: Case of Perforation of the Appendix Vermiformis—Death from Peritonitis. Am. Jour. Med. Sc., vol. xxii, pp. 127-131, May, 1838.
- ⁸¹ Hancock: Disease of the Appendix Cæci Cured by Operation. Lancet, vol. ii, pp. 380–382, September 30, 1848.
- ** Hansen, P. N.: Om Behandlingem af gangrense Hernier. Nord. Tidsskr. f. Terapi, vol. iv, pp. 202-209, 1905-1906.
- ⁸³ Harrenstein, R. J.: Appendectomy and the Ileocæcal Gland. Nederl. Tijdschr. v. Geneesk., vol. i, p. 2652, June 19, 1926; abstr. in: J. A. M. A., vol. lxxxvii, p. 454, August 7, 1926.
- ⁸⁴ Harrenstein, R. J.: Eine notwendige Korrektur der herrschenden Auffassungen über die Bedeutung der Appendixhäsionen in dem Krankheitsbilde der chronischen Appendicitis. Beitr. z. klin. Chir., vol. cxxxix, pp. 533-543, 1927.
- 85 Hathaway, F. J.: The So-called Chronic Appendix. Practitioner, vol. cxvii, pp. 240–251, October, 1926.
- Hedinger, E.: Kongenitale Divertikelbildung im Processus vermiformis. Virchow's Arch. f. path. Anat. u. Physiol., vol. clxxviii, pp. 25-43, October, 1904.
- ⁸⁷ Herrick, F. C.: Discussion. Ohio State Med. Jour., vol. xix, pp. 846–848, December, 1923.
- 88 Hertzler, A. E.: An Inquiry into the Nature of Chronic Appendicitis. Am. Jour. Obst. and Gynec., vol. xi, pp. 155-170, February, 1926.
- ⁸⁰ Heyd, C. G.: Review of Some Recent Work on Surgical Physiology of the Gastrointestinal Tract with Special Reference to the So-called Chronic Appendicitis. Ohio State Med. Jour., vol. xx, pp. 749-756, December, 1924.
- ⁹⁰ Horsley, J. S.: Unperforated Ulcers of Terminal Ileum, Symptomatically Simulating Appendicitis. J. A. M. A., vol. 1xxxv, pp. 863-867, September 19, 1925.
- ⁹¹ Husson, and Dance: Répertoire général d'anatomie et de physiologie pathologiques. Paris, Boiste, vol. iv, p. 154, 1827.
- ⁹² Illoway, H.: Chronic Appendicitis and Hyperacidity of the Gastric Juice. New York Med. Jour., vol. xcviii, pp. 162-168, July; pp. 224-227, August, 1913.
- ⁶⁰ Kelly, H. A., and Hurdon, E.: The Vermiform Appendix and Its Diseases. W. B. Saunders Company, 827 pp., Philadelphia, 1905.
- Lewis, G.: A Statistical Contribution to Our Knowledge of Abscess and Other Diseases Consequent Upon the Lodgment of Foreign Bodies in the Appendix Vermiformis, with a Table of Forty Cases. New York Jour. Med., vol. iii, pp. 328-353, 1856.
- ⁶⁶ Lichty, J. A.: Chronic Appendicitis. J. A. M. A., vol. 1xxix, pp. 887–893, September 9, 1922.
- Lichty, J. A.: Appendicitis. In: Tice's Practice of Medicine, W. F. Prior Company, vol. vii, pp. 623-638, Hagerstown, 1927.
- 97 Lockwood, C. B. and Rolleston, H. D.: On the Fossæ Around the Cæcum, and the

Position of the Vermiform Appendix, with Special Reference to Retroperitoneal Hernia. Jour. Anat. and Physiol., vol. xxvi, pp. 130-148, October, 1891.

⁸⁸ Lockwood, G. R.: Diseases of the Stomach. Lea and Febiger, 624 pp., Philadel-phia, 1913.

¹⁰⁰ Louyer-Villermay: Observations pour servir à l'histoire des inflammations de l'appendice du cœcum. Arch. gén. de méd., vol. v, pp. 246–250, 1824.

Maale, C. U.: Histopatologiske studier over processus vermiformis. Jacob Lund, 266 pp., Kobenhaven, 1908.

MacCarty, W. C.: Classification of Appendicitis and the Relation of Chronic Appendicitis to Obliteration of the Lumen, Carcinoma, and Disturbances in the Gastrohepatico-duodeno-pancreatic Physiologic System. J. A. M. A., vol. lv, pp. 488-492, August 6, 1910.

MacCarty, W. C.: The Pathology of the Gall-bladder and Some Associated Lesions: A Study of Specimens from 365 Cholecystectomies. Annals of Surgery, vol. li, pp. 651-669, May, 1910.

MacCarty, W. C., and McGrath, B. F.: Clinical and Pathological Significance of Obliteration, Carcinoma, and Diverticulum of the Appendix; Deductions from an Examination of 5,000 Specimens, with a Comparative Study of the Pathology and Clinical Histories in 2,000 Cases. Surg., Gynec. and Obstet., vol. xii, pp. 211–220, March, 1911.

MacCarty, W. C., and McGrath, B. F.: The Frequency of Carcinoma of the Appendix: A Report of Forty Cases (.44 Per Cent) in 8,039 Specimens. Annals of Surgery, vol. lix, pp. 675-678, May, 1914.

¹⁰⁵ MacLaren, Archibald: Chronic Appendicitis and Its Relation to Visceroptosis. Annals of Surgery, vol. lxiv, pp. 579-584, November, 1916.

¹⁰⁵ Mallory, F. B.: The Principles of Pathologic Histology. W. B. Saunders Company, pp. 486–487, Philadelphia, 1929.

Masson, P.: Les lésions nerveuses de l'appendicite chronique. Compt.-rend. Acad. d. sc., vol. clxxiii, pp. 262-264, 1921.

Masson, P.: Le néuromes sympathiques de l'appendicite oblitérante. Lyon chir., vol. xviii, pp. 281-299, May-June, 1921.

Masson, P.: Les lésions du plexus nerveux périglandulaire dans l'appendicite chronique. Bull. et mém. Soc. méd. d. hôp. de Paris, vol. xlvi, pp. 956-969, 1922.

Masson, P.: Appendicite neurogène et carcinoides. Ann. d'anat. pathol. méd.-chir., vol. i, pp. 3-59, January, 1924.

Masson, P.: Carcinoids (Argentaffin-cell Tumor) and Nerve Hyperplasia of the Appendicular Mucosa. Am. Jour. Path., vol. iv, pp. 181-211, May, 1928.

Mathieu, A.: La douleur paroxystique dans le syndrome ulcéropylorique. Rev. gén. de clin. et de thérap., vol. xxviii, pp. 436-438, 1914.

¹¹³ Matterstock, G. K.: Perityphlitis. In: Gerhardt, Carl: Handbuch der Kinderkrankheiten, vol. iv, pp. 893-928, 1880.

¹¹⁴ Mayo, C. H.: The Surgical Physiology of the Lymphatic System. Tr. South. Surg. and Gynec. Assn., vol. xvi, pp. 442–452, 1903.

¹¹⁵ Mayo, C. H.: The Appendix in Relation to, or as the Cause of, Other Abdominal Diseases. J. A. M. A., vol. lxxxiii, pp. 592–593, August 23, 1924.

^{115a} Mayo, W. J.: Surgery of the Large Intestine: with a Review of One Hundred Resections. Annals of Surgery, vol. 1, pp. 200-228, July, 1909.

Mayo, W. J.: The Ileocæcal Orifice and Its Bearing on Chronic Constipation, with Report of Two Cases Relieved by Operation. Annals of Surgery, vol. xxxii, pp. 364-368, September, 1999.

Mayo, W. J.: Resection of the First Portion of the Large Intestine and the Resulting Effect on Its Function. J. A. M. A., vol. 1xiii, pp. 446-449, August 8, 1914.

Mayo, W. J.: Chronic Appendicitis. Editorial. Surg., Gynec. and Obstet., vol. xlii, pp. 717-718, May, 1926.

13

- ²¹⁰ Mayo, W. J.: The Enlarged Spleen. South. Med. Jour., vol. xxi, pp. 13-16, January, 1928.
- ¹²⁰ McBurney, Charles: Experience with Early Operative Interference in Cases of Disease of the Vermiform Appendix. New York Med. Jour., vol. 1, pp. 676-684, December 21, 1889.
- ¹²¹ McCarrison, Robert: Faulty Food in Relation to Gastro-intestinal Disorder. J. A. M. A., vol. lxxviii, pp. 1–8, January 7, 1922.
- ¹⁹² Melchior, and Loser: Quoted by Rost, F.: The Pathological Physiology of Surgical Diseases. P. Blakiston's Son and Company, 263 pp., Philadelphia, 1923.
- Melier, F.: Mémoire et observations sur quelques maladies de l'appendice cæcale. Jour. gén. de méd., de chir. et de pharm., vol. c, pp. 317-345, 1827.
- Menière, P.: Mémoire sur des tumeurs phlegmoneuses occupant la fosse iliaque droite. Arch. gén. de méd., vol. xvii, pp. 188-218, June; pp. 513-532, August, 1828.
- Menon, T. B.: Some Pathological Aspects of Chronic Appendicitis. I. The Lymphoid Tissue of the Appendix. Indian Jour. Med. Res., vol. xvi, pp. 656-660, January, 1929; II. Eosinophile Infiltration of the Appendix. Indian Jour. Med. Res., vol. xvi, pp. 661-663, January, 1929.
- ¹²⁰ Menon, T. B.: Some Pathological Aspects of Chronic Appendicitis. III. The Histological Diagnosis. Indian Jour. Med. Res., vol. xvi, pp. 1019–1022, April, 1929.
- Mertens, V. E.: Falsche Divertikel der Flexura sigmoidea und des Processus vermiformis. Mitt. a. d. Grenzgeb. d. Med. u. Chir., vol. ix, pp. 743-761, 1902.
- ¹²⁸ Mestivier: Observation sur une tumeur située proche la région ombilicale, du côté droit, occasionnée par une grosse épingle trouvée dans l'appendice vermiculaire du cæcum. Jour. de méd., de chir., et de pharm., vol. x, p. 441, 1759.
- Montais, Camille: Contribution à l'étude de l'appendicite chronique d'emblée (phénomènes dyspeptiques). Paris, 417 pp., 1900.
- ¹³⁰ Morris, R. T.: Appendicitis. Internat. Jour. Surg., vol. xi, p. 238, 1898.
- Morris, R. T.: Five Kinds of Chronic Appendicitis. Am. Jour. Obst. and Gynec., vol. xi, pp. 180-183, February, 1926.
- Moynihan, B. G. A.: Remarks on Appendix Dyspepsia. Brit. Med. Jour., vol. i, pp. 241-244, January 29, 1910.
- Moynihan, B. G. A.: Duodenal Ulcer. W. B. Saunders Co., Ed. 2, 486 pp., Phila., 1912.
- Moynihan, B. G. A.: Gastric Ulcer and Its Treatment. Med. Rec., vol. xcix, pp. 903-910, May 28, 1921.
- ¹³⁶ Mundt, R.: Über Veränderungen der Muskelward des Wurmfortsatzes. Pathol.anat Arbeiten. Joh.: Orth z. Prof.-Jubil., pp. 453-470, Berlin, 1903.
- Murphy, J. B.: Acute Appendicitis. Surg. Clin., John B. Murphy, vol. ii, pp. 107-117, Chicago, November, 1913.
- Mummery, J. P. L.: Diseases of the Colon and Their Surgical Treatment. William Wood and Co., 322 pp., New York, 1910.
- Nicholson, H. G.: Plea for the Normal Appendix; Its Physiology as Viewed from a Surgical Point. West Virginia Med. Jour., vol. xv, p. 244, January, 1921.
- Nicolle, Charles and Conseil, E.: Vaccinations préventives par voie digestive chez l'homme dans la dysenterie bacillaire et la fièvre méditérranéenne. Ann. de l'Inst. Pasteur, vol. xxxvi, pp. 579-613, August, 1922.
- Ochsner, A. J.: Appendicitis (Summary). Jour. Michigan Med. Soc., vol. iii, pp. 371-378, 1904.
- ¹⁴¹ O'Neil, R. F.: A Plea for the Early Recognition of the Symptoms of Urologic Lesions. J. A. M. A., vol. lxxvii, pp. 417-421, August 6, 1921.
- Parker, Willard: An Operation for Abscess of the Appendix Vermiformis Caci. Med. Rec., vol. ii, pp. 25-27, March 15, 1867.
- ¹⁴³ Parkinson, James: Case of Diseased Appendix Vermiformis. Med. Chir. Tr., vol. iii, pp. 57-58, London, 1812.
- Paterson, H. J.: Jejunal and Gastrojejunal Ulcer Following Gastrojejunostomy. John Bale, Sons and Danielson, Ltd., 72 pp., London, 1909.

HISTORIC PHASES OF APPENDICITIS

- Paterson, H. J.: Appendicular Gastralgia, or the Appendix as a Cause of Gastric Symptoms. Proc. Roy. Soc. Med. (Surg. Sec.), vol. iii, pp. 187-208, 1910.
- Paterson, H. J.: The Surgery of the Stomach. James Nisbet and Co., Ltd., 312 pp., London, 1913.
- ¹⁴⁷ Paus, N.: [Difficulties in Diagnosing Appendicitis.] Norsk. Mag. f. Laegevidensk., vol. lxxxv, pp. 762–764, 1924.
- Payr, E.: Über die sogennante primär-chronische, klinischanfallsfreie Appendicitis. Die Gelegenheitsappendektomie. Deutsch. Ztschr. f. Chir., vol. cc, pp. 307–363, March, 1927.
- ¹⁴⁰ Pitzman, Marsh: "No Surgical Appendicitis Without Organic Stricture." Annals of Surgery, vol. lxxi, pp. 149-154, February, 1920.
- Price, J.: Appendicitis, Its Surgical Treatment. Med. Herald, vol. xix, pp. 81–88, 1990.
- ¹⁵¹ Ramond, Félix, and Parturier, G.: Cholécystite et appendicite chroniques. Presse méd., vol. i, pp. 710-711, June 5, 1926.
- Rastouil, A.: Appendicite chronique. Imp. de la Faculté de Méd., n. 223, 95 pp., Paris, 1901.
- ¹⁵⁸ Reischauer, F.: Appendicitis und vegetatives Nerwensystem. Hat Ricker recht? Beitr. z. klin. Chir., vol. cxlviii, pp. 283–300, December, 1929.
- Ribbert, H.: Beiträge zur normalen und pathologischen Anatomie des Wurmfortsatzens. Virchow's Arch. f. path. Anat. u. Physiol., vol. cxxxii, pp. 66-90, April 22, 1893.
- ¹⁵⁶ Richards, W.: A Case of Death from Ulceration of the Appendix Vermiformis. West Jour. Med. and Phys. Sc., vol. xi, pp. 376-379, 1838.
- Richardson, M. H. and Brewster, G. W. W.: Appendicitis, Remarks Based upon a Personal Experience of 750 Cases; Including 150 Consecutive Cases Successfully Operated upon "In the Interval." Boston Med. and Surg. Jour., vol. cxxxix, pp. 25–28, July 7, 1898.
- Rogalsky, B. A.: Involution of the Appendix Vermiformis. Vestnik. khir., vol. xii, pp. 38-72, 1928.
- ¹⁶⁸ Rosenow, E. C.: The Bacteriology of Appendicitis and Its Production by Intravenous Injection of Streptococci and Colon Bacilli. Jour. Infect. Dis., vol. xvi, pp. 240–268, March, 1915.
- ¹⁵⁰ Rosenow, E. C.: Elective Localization of Streptococci. J. A. M. A., vol. lxv, pp. 1687–1691, November 13, 1915.
- Rosenow, E. C.: Focal Infection and Elective Localization of Bacteria in Appendicitis, Ulcer of the Stomach, Cholecystitis, and Pancreatitis. Surg., Gynec. and Obstet., vol. xxxiii, pp. 19-26, July, 1921.
- ¹⁶¹ Rosenow, E. C.: Serologic Specificity of Streptococci Having Elective Localizing Power as Isolated in Various Diseases of Man. Jour. Infect. Dis., vol. xlv, pp. 331–359, November, 1929.
- Poux, Jacques: De l'emploi des rayons X avant l'appendicectomie. Clin. Chir. de l'Université de Lausanne, 49 pp., 1913.
- Sands, H. B.: Account of a Case in Which Recovery Took Place After Laparotomy Had Been Performed for Septic Peritonitis Due to a Perforation of the Vermiform Appendix, with Remarks Upon This and Allied Diseases. New York Med. Jour., vol. xlvii, pp. 107-205, February 25, 1888.
- ¹⁶⁴ Sanes, K. I.: Ureteral Obstruction. J. A. M. A., vol. 1xxviii, pp. 475-477, February 18, 1922.
- Savkoff, N. M.: Results of Appendectomy in Chronic Appendicitis. Rpssk. Klin., vol. x, pp. 366–385, September–October, 1928.
- 186 Schnitzler, J.: So-called Chronic Appendicitis. Wien. klin. Wchnschr., vol. xxxviii, pp. 1-24, January, 1925.

- ¹⁶⁷ Schoemaker, Jan: The Red Stomach. Surg., Gynec. and Obstet., vol. xl, pp. 305–309, March, 1925.
- 168 Seedham, and Green: The Frequency with Which Chronic Inflammatory Troubles in the Appendix Give Rise to Typical Symptoms of Gastric and Duodenal Ulcer. Birmingham Med. Rev., pp. 20–22, 1914.
- Shapiro, Frank: Pseudo-appendicitis in Children. Med. Jour. and Rec., vol. cxxvii, pp. 383-384, April 4, 1928.
- ¹²⁰ Sherren, James: Appendicitis. Brit. Med. Jour., vol. i, pp. 727–730, April 18, 1925.
- ¹⁷¹ Shutt, C. H.: Pericolic Adhesions as a Factor in Diagnosis of Chronic Appendicitis and as a Factor in the Failure to Obtain Relief by Removal of the Appendix in Some Cases. Jour. Missouri Med. Assn., vol. xxii, pp. 249-253, July, 1925.
- ¹⁷² Singer, Charles: On the Secretory Activity of the Stomach in Chronic Appendicitis with Gastric Symptoms. Lancet, vol. ii, pp. 1711–1713, December 21, 1912.
- ¹⁷⁸ Siredey, A.: Étude clinique sur quelques formes frustes de l'appendicite chronique. Bull. et. mém. soc. méd. d. hôp. de Paris, vol. xx, pp. 1274–1302, November 20, 1903.
- Stanton, E. McD.: An Analysis of the Deaths Occurring in the Course of 1,573 Surgical Operations. Albany Med. Ann., vol. xxxv, pp. 432-449, June, 1914.
- Stanton, E. McD.: Chronic Appendicitis: A Study of Post-operative End-results. New York Med. Jour., vol. cx, pp. 406-409, September 6, 1919.
- Trotter, Wilfred: Discussion on Chronic Appendicitis. I. Symptomatology and Diagnosis. Brit. Med. Jour., vol. ii, pp. 1063–1065, December 10, 1927.
- Vasselle, P., and Parturier, G.: Duodenal Ulcer. Progrès méd., vol. xxxvi, p. 495,
 October, 1921.
- Visani, Carlo: Sintomia patologica dell'addome destro (appendicite, colecistite, ulcera gastrica e duodenale) & frequenza. Meccanismo patrogenetico. Clin. Med. d. R. Universita' Di Firenze, 116 pp., 1927.
- Volz, A.: Ueber die Verschwärung und Perforation des Processus vermiformis, bedingt durch fremde Körper. Arch. f. d. ges. Med., vol. iv, pp. 305–338, 1843.
- Volz, Adolph: Die durch Kothsteine bedingte Durchbohrung des Wurmfortsatzes.
 C. F. Müller, 121 pp., Carlsruhe, 1846.
- ¹⁶⁵ Waltner, J.: Sur une forme d'appendicite chronique à evolution latente sans aucune crise aigue. Cong. franç. de chir., 1908.
- Walton, A. J.: Discussion on Chronic Appendicitis. IV. Etiology and Sequels of Chronic Appendicitis. Brit. Med. Jour., vol. ii, pp. 1068-1070, December 10, 1927.
- Walton, A. J.: Chronic Appendicitis in Children. Lancet, vol. i, pp. 595–596, March 24, 1928.
- Weber, Leonard: Abscess of the Appendix Vermiformis. New York Med. Jour., vol. xiv, pp. 142-163, August, 1871.
- Wegeler: Historia enteritiditis malignae et singularis calculosi concrementi. Jour. de med., de chir., et de pharmacol., vol. xxviii, p. 384, 1813.
- 153 Weinberg, M.: Unpublished data.
- Whiteford, C. H.: Acute Appendicitis, Practical Points from a Twenty-five Years' Experience. Harrison and Sons, 72 pp., London, 1917.
- Whiteford, C. H.: The Chronic Appendix. Practitioner, vol. cix, pp. 155-165, August, 1922.
- ¹⁵⁰ Williams, J. T., and Slater, Robert: The Condition of the Appendix in Five Hundred Laparotomies on Patients Presenting No Symptoms of Appendicitis. Annals of Surgery, vol. Ixx, pp. 535-538, November, 1919.
- With, C.: Peritonitis appendicularis eller den ved ulceration og perforation af appendix ileocoecalis fremkaldte peritonites. Nord. med. Ark., vol. xii, p. 1722, 1880; Transl. (abstr.) London Med. Rec., n. s., vol. viii, pp. 213-215, 1880.
- Woodbury, Frank: Case of Exploratory Laparotomy Followed by Appropriate Remedial Operation. Tr. Coll. Phys. and Surg., vol. ix, pp. 183-193, Philadelphia, June, 1887.

THE CHANGING PICTURE OF APPENDICITIS IN ADULTS

A REVIEW OF 1,000 CASES

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This study was undertaken in the attempt to offer evidence of the present picture of appendicitis, changed in some respects when compared to that of some years ago. We have noted an increase in the mortality rate of the disease in late years, and have raised the question as to the reason for this. It is granted that appendicitis can be cured usually if the proper operation is performed sufficiently early in the course of the infection. Therefore, it may be possible that the faulty or late diagnoses with consequent increased mortality are due to some cardinal changes in the picture of the disease. To form a basic idea of the older understanding of appendicitis and to be able to compare it with our findings in this series it is necessary first to review the standard picture of the disease as accepted in the past.

Literature.—A summary of the outstanding symptoms and signs of appendicitis of a somewhat earlier day may be established by transcribing the opinions of a group of acknowledged authorities. It is manifestly impossible to quote from all contributors to the voluminous literature of appendiceal infection; hence, we have chosen to present the opinions of a group of men whose words carry great weight when presented to the profession. A very important point is that their works are strictly modern, with one or two exceptions. In many instances, they are authors of books which have passed through several editions, and in which the fact that the picture of appendicitis is somewhat changed from that of a decade or more ago is not stressed.

For example, Heyd and Killian,1 in 1924, emphasize in appendicitis, pain, nausea, vomiting, abdominal sensibility and irritable colon, with either diarrhoea or constipation; Cope^a remarks the acute type of infection, mentions the consistent presence of pain, nausea, vomiting, constipation, fever and inconstant rigidity and distension; of the same type, Royster3 speaks of pain, vomiting, tenderness, rigidity, fever, and says that constipation is usual; Kelley,4 in his monumental text of 1911, says that constipation is one of the most constant symptoms in chronic appendicitis and that the majority of patients with the acute form are constipated. He also stresses abdominal distension in addition to the other signs and symptoms already noted above; Graham⁶ emphasizes the common evidences and notes the bowels to be constipated ordinarily. He also remarks distension in delayed cases; Cecil⁶ says that pain, tenderness, rigidity, fever and leucocytosis are important and, interestingly enough, that the majority of patients either are constipated or have normal bowel elimination; the outstanding findings according to Wakeley and Hunter are pain, fever, nausea, vomiting and constipation; Lewis emphasizes the usual symptoms and signs and states that constipation is usual if no cathartic has been given; and, finally, McCrae⁹ says that constipation is the rule along with pain, fever, nausea, vomiting and tenderness.

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Our findings have been at some variance with those noted above and it is because of this fact that they are presented. A thousand cases of appendicitis do not form a large series as this disease is often reported, but the evidence presented by such a number must be of some value.

Report of this series.—This review is of the histories of 1,000 consecutive patients suffering with appendicitis, all of whom were treated surgically. All have detailed pathologic reports which formed the basis for our grouping.

The case histories chosen were of those patients who presented no major complications of their appendiceal infection, with few exceptions. We have weeded out most of those with some accompanying serious disorder, for we wished to get an accurate picture of the disease of the appendix alone, in so far as we were able. Incomplete histories were considered only occasionally, *i.e.*, if they presented some especially important points. No effort was made to consider the finer details of the diagnosis, such as duration of pain, number of vomiting attacks, cutaneous hyperæsthesia, *etc.*, for these findings were inconsistently noted. Instead, only the frequency of the major outstanding symptoms and signs have been recorded. The series goes back from the present to include some histories of 1924.

Grouping-type.—Manifestly the 1,000 cases had to be grouped according to some plan with regard to the type represented. They were grouped strictly as to the pathologic findings which accorded closely with the clinical picture presented, into chronic, acute simple, and acute suppurative types, corresponding to clinical chronic, acute and suppurative appendicitis. Dr. B. S. Kline, of the Pathological Department, describes the microscopic features of these types as follows: Acute simple appendicitis presents microscopically a picture of varying degrees of inflammation and ædema of the organ with serum, fibrin and leucocytes present. The chronic type is that with some increase in connective tissue, the cell infiltration being of predominately mononuclear character, all of varying degree. Acute suppurative changes show an intense infiltration by pus-cells of one or more coats of the organ with destruction of fixed tissue. If gangrene is present, not only is the inflammation more severe, but also there is necrosis of fixed tissue and exudate. Some of these show rupture, and some do not. There is always more or less periappendicitis present.

Grouping-age.—Inasmuch as the picture of appendicitis varies greatly with the age of the patients, it was necessary to group them in some way for convenience of study. Therefore, we began with the earliest age of adulthood, 13 years, and made the first group of individuals of the ages 13–20 inclusive and after that divided them into decades.

Tables.—The tables are made up almost entirely of percentages and averages. This simplifies the recording of the pictures presented and conserves much space. In the tables, in accord with statements above, we have noted only the outstanding items: type, age group, sex, the symptoms: pain, nausea, vomiting, bowel condition, and the signs: rigidity, distension, area of maximum tenderness, pulse, temperature, leucocyte count. The preponderance of

APPENDICITIS CHANGING IN ADULTS

female patients is not remarkable in this hospital which, though of the general type, attracts a majority of the female sex.

Our findings are summarized in the tables.

Discussion of acute suppurative group.—This type, called clinically suppurative appendicitis, is represented by the severe infections. As would be expected, this is the smallest group. These cases represent the "delayed" infections in which there is present, at the very least, a suppurative process with gangrene frequently grafted on it and often an abscess present. Many of these appendices were found to be ruptured. Drainage was the common surgical procedure. Our findings are of considerable interest even in this severe type. In the following respects our tabulations agree entirely with those of the literature: The presence of abdominal pain in practically every patient, with nausea and vomiting in the majority; the physical findings of rigidity of the abdominal muscles in most instances; in the distribution of the area of maximum tenderness as proportioned between the McBurney region and the right lower quadrant of the abdomen; in the typical average findings of pulse, temperature and leucocyte count at time of entrance to the hospital. However, there are some points in which our results do not agree with those commonly quoted. In this regard we may mention that constipation was present in a comparatively small minority of patients with the exception of the oldest ones. Diarrhœa was present in a very small percentage. In the various age groups, from 9 per cent. to 35 per cent. gave us a history of having taken a cathartic, which, of course, may have been responsible for the serious findings in their appendices. It is worth while to call attention to the comparatively high percentage of individuals whom we found to have entirely normal bowel elimination, the figures varying from 34 per cent. to 44 per cent. in the different age groups. We must not overlook the fact that only a small number of our patients suffered with abdominal distension, the figures averaging about 20 per cent. for all age groups. To summarize: Our tabulations show a comparatively small number of patients suffering with constipation and abdominal distension and very few with diarrhoea, and, conversely, a rather large proportion with normal bowel elimination, when we bear in mind the extremely acute process with which they were suffering.

Discussion of acute simple group.—Clinically, this type is called "acute appendicitis." The process is relatively less severe than that of the suppurative group as illustrated by our averages of pulse, temperature and leucocyte count results which conform to the usual figures. In our percentages, pain, nausea, vomiting, rigidity and distribution of tenderness appear in about the standard proportions. However, constipation was present in only about one-third of the patients, diarrhea was unusual, and almost one-half of the patients reported their elimination through the bowels to be normal. The percentage of those in which abdominal distension was found was very low in all age groups. Briefly, again we found that constipation is not by any means the usual picture and distension is unusual, whereas

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TABLE I

about one-half of the patients were not suffering with any abnormality of bowel emptying.

Discussion of chronic group.—Our findings of pain, nausea and vomiting in attacks, location of tenderness, the presence of rigidity and distension, with average pulse, temperature and leucocyte figures are about what we would expect from the classical description of this disease. The complaint of distension in the acute phases of their chronic inflammatory disease would place these patients in about the same class as those whom we encountered with acute appendicitis at time of entrance to the hospital. At least it seems fair to conclude thus from our percentages. It has been previously maintained by various authors that chronic appendicitis represents merely a resting stage of the appendix between attacks of sub-acute or acute inflammation. This fact is further borne out by our findings regarding bowel condition in this chronic group in which constipation was present in about the same proportion as in the acute group, and diarrhoea was unusual. It should be noted, however, that less than one-half the patients suffered with constipation either in or between attacks, whereas a majority of them had normal bowel elimination. Our summary of this group must be similar to that of the acute group.

Summary.—One thousand case histories of adult* patients suffering with appendicitis have been analyzed in an effort to determine with what consistency were present the classical symptoms and signs of the disease as stated by authoritative writers in past publications. A number of these authors were quoted together with their opinions as to the outstanding features of the infection. For purposes of review, our series was separated into three divisions based on pathological findings, *i.e.*, the acute suppurative, acute simple and chronic groups. Clinically, these types are called suppurative appendicitis, acute appendicitis and chronic appendicitis.

It is difficult to summarize at one time the findings of the three main groups. The pictures are so variable that it is undoubtedly better to consider them separately. In the main, our percentages and averages accord with the standard as far as the presence of abdominal pain, the occurrence of nausea, vomiting, and rigidity, the distribution of tenderness between the McBurney area and the right lower quadrant, and the findings of pulse, temperature, and leucocyte count at time of admission, are concerned. In regard to the presence of constipation, diarrhea and normal bowel elimination, with abdominal distension, our findings are distinctly at variance. These evidences of appendicitis were present in a much smaller proportion of our patients than might be expected from the classical descriptions of various authors.

In our experience, typical major symptoms of appendicitis, varying from the chronic through the acute to the suppurative forms, include: (1) Pain in the abdomen at the time of the attack in almost every instance. (2) Nausea and vomiting present fairly consistently. (3) Rigidity of the abdominal

^{*} See also "Appendicitis in Children under Thirteen Years of Age" by the same author, Ohio State Medical Journal, vol. xxvii, No. 6, p. 461, June 1, 1931.

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muscles more often found in the acute forms. (4) Maximum tenderness about evenly distributed between the McBurney area and the right lower quadrant. (5) Variable increase in pulse rate, temperature and leucocytosis depending on the severity of the infection. (6) Normal bowel elimination in about half the patients. (7) Constipation in the minority. (8) Abdominal distension in a small percentage. (9) Diarrhoea in only the occasional case.

Conclusions.—From a study of 1,000 case histories of appendicitis in adults, with reference to the major symptoms and signs, it seems justifiable to conclude that the present-day picture of the disease is not exactly the same as was the picture of several years ago; that, whereas abdominal pain, nausea, vomiting, muscular rigidity, abdominal tenderness and elevation of pulse, temperature and leucocyte count occur in about the same proportions as formerly, at present there is a lower incidence of constipation, diarrhæa and abdominal distension in these patients; and that there need be no hesitancy in the diagnosis of appendicitis in individuals who present the cardinal symptoms and signs even though the abdomen shows no distension and the state of intestinal elimination is normal.

REFERENCES

¹ Heyd, C. G., and Killian, J. A.: The Liver and Its Relation to Chronic Abdominal Infection. P. 59, 1924.

² Cope, Z.: The Early Diagnosis of the Acute Abdomen. Oxford University Press, p. 53, 1928.

⁸ Royster, H. A.: Appendicitis. P. 93, 1927.

Kelley, H. A.: The Vermiform Appendix. P. 386, 1911.

<sup>Graham, E. A.: Surgical Diagnosis. P. 636, 1930.
Cecil, R. L.: Text Book of Medicine. P. 748, 1930.</sup>

Wakeley and Hunter: Rose and Carless' Manual of Surgery. P. 1185, 1030.

^{*}Lewis, Dean: Practice of Surgery. Vol. vii, p. 10, 1929.

⁶ McCrae, Thomas: Osler's Principles and Practice of Medicine. P. 529, 1930.

APPENDICITIS CHRONIC*

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FROM THE HENRY FORD HOSPITAL

THE number of papers on the subject of appendicitis is enormous. The records of the "Index Medicus" and the quarterly cumulative "Index Medicus" show that the total number reaches 9,273, while there are approximately 662 on chronic appendicitis.

Our excuse is, that as the weight of opinion against any clinical entity of symptoms due to chronic appendicitis increases, the death rate from appendicitis has increased and that recently we have taken an inventory of our own experiences in the operating rooms and pathological laboratories of the Henry Ford Hospital. The pendulum of opinion against the very existence of such a thing as chronic inflammation of the appendix has swung so far that it has been many years since we, except on rare occasions, have scheduled operations for chronic appendicitis in our hospital excepting when there has been definite history of acute attacks. Many papers have been published which would make it appear that a chronic inflammatory reaction in the appendix is impossible. We all know that chronic inflammatory reactions (meaning those extending over a longer period of time than acute signifies) do persist in the pelvis, around ulcers, in the bronchi, in the tonsils, around teeth and the paranasal sinuses, in bone, etc. It would seem strangely incongruous, therefore, if chronic inflammation could not occur in the appendix in view of its anatomical structure which is so particularly suited for the existence of such a condition. In view of the opinion of the majority of pathologists that there is a definite entity of chronic appendicitis, is it possible that this pendulum of opinion against any such clinical diagnosis of chronic appendicitis has swung far enough? Should we study this subject further, especially in view of the fact that no advances in twenty-five years or more have been made in the diagnosis and treatment of appendicitis, acute or chronic, while great advances have been made in the diagnosis and treatment of gall-bladder disease?

Aschoff¹ believes "that the frequency of acute irritative lesions in the appendix is under-rated and that destructive lesions in the appendix are the result of these attacks predisposing the appendix to new infections. These slowly healing acute attacks, these ever-recurring acute inflammations, might be called chronic appendicitis but they would nevertheless be secondary chronic processes—effects of a primary acute irritation."

Whether we think of a continuous process, as the term chronic implies, or in the sense that Aschoff regards it, makes but little difference in the surgical consideration.

^{*} Read before the Southern Surgical Association, December 10, 1930.

ROY D. McCLURE

The subject of appendicitis in general, as shown by the increasing death rate from appendicitis in the United States, is of paramount importance. The study of it suggests that the medical profession is not measuring up as it might reasonably be expected to do in meeting this emergency in the way of public education and in improved early diagnosis and surgical treatment.

Table I shows the increasing death rate, an appreciation of which, may arouse further interest in the subject of chronic appendicitis, as a possible key to a situation which should be preventable, for parallel to this increasing death rate has run the decrying of chronic appendicitis as an entity.

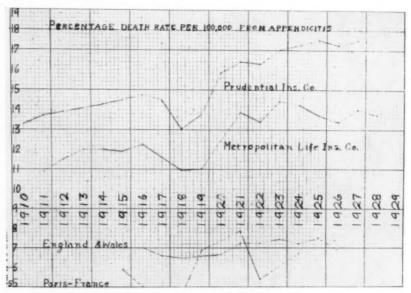


Table I.—Percentage death rate per 100,000. (Hoffman, J. L., Appendicitis Record for 1928.)

The 17,687 deaths from appendicitis last year in the United States should give medical men food for thought, for these deaths are largely among citizens in the productive years of life and are preventable, we feel, if every individual had prompt and perfect care.

Hoffman⁷ in the "Appendicitis Record for 1928" shows in the following international statistics the wide variation of appendicitis mortality throughout the world.

TABLE II
INTERNATIONAL STATISTICS OF APPENDICITIS

Rates per 100,000

	Year	Rate
United States	1927	15
Switzerland	1926	11
Scotland	1926	10
Sweden	1924	9
Belgium	1925	7
England and Wales		7

CHRONIC APPENDICITIS

	Year	Rate
North Ireland	1926	7
Germany	1026	7
Norway	1924	7
Irish Free State	1926	6
Czechoslovakia	1926	4
Esthonia	1925	4
Netherlands	1926	4
France	1925	3
Italy	1925	3
Spain	1926	3
Greece	1926	2
Lithuania	1926	2

Our country ranks highest in death rate, whereas some countries do not have appendicitis. A few years ago, Doctor McCarrison, of the British Army in India, while visiting our hospital, stated that in fifteen years' experience in a region of the Himalaya Mountains he had never seen or heard of a single case of appendicitis among the population of about 15,000,000 people. Doctor McCarrison's only explanation was that in his district in India they consumed no sugar excepting as it occurs in natural foods and he pointed to the tremendous increase per capita of the use of sugar in the United States. He also mentioned that there were no canned goods used in that country.

Dr. Paul Harrison, a man of outstanding medical ability who has had twenty busy years in medical missionary work, states that in twenty continuous years' experience in Arabia he has seen and operated upon only one case of appendicitis. One physician asked him why they did not have appendicitis in Arabia. With a rare twinkle in his eye he said: "Do you not rather feel it is your duty to tell us why you have appendicitis in the United States?"

Dr. Adrian Taylor, however, reports to me that appendicitis is a common disease in those parts of China with which he is familiar.

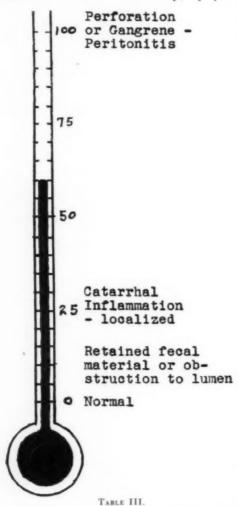
As to whether there exists in the natives of these countries, where even acute appendicitis is non-existent, a chronic appendicitis such as described by our pathologists would be of interest, and we are now endeavoring to obtain from these countries some appendices removed during the course of other operations for further study.

That there is a definite entity from a pathological viewpoint of chronic appendicitis seems fairly well agreed upon in the countries where acute appendicitis is prevalent. That this condition is more than a senile change seems probable. If the term "chronic inflammation" were limited to infections of tuberculosis, syphilis or actinomycosis we would have to find a new term for the type of appendicitis we are now discussing—perhaps we would call it "subacute appendicitis." Whether this wide group of people with chronic appendicitis (pathological diagnosis) serves as the foundation for mounting mortality from acute exacerbations should now be determined.

Osler twenty-five years ago wrote: "The lumen of the appendix forms

a sort of test-tube, in which the fæces lodge and are with difficulty discharged so that the mucosa is liable to injury from retention of the secretions or from the presence of inspissated fæces or occasional foreign bodies."

It seems very logical to believe that local irritation in the appendix is followed by inflammation unless that irritation is relieved by natural peristalsis or if not in this way, by perforation, pus-appendix, gangrene or



whatnot. The degree of inflammation depends on the length of time the irritating matter is present and the virulence of the bacteria. (Aschoff believes there may be specific organisms.) Why there should be retention of inspissated material in the appendix in this country and not in parts of India and Arabia might depend on several factors, i.e., food, bowel habits, nervous tension, etc. The food factors are peculiar in that the natives have more roughage in their food, no refined sugar, no canned goods, etc. They do not conform to the more refined habits of civilization regarding the passage of gas and the time and place for bowel movements. They are under none of the nervous tension and worry of our present age, a tension which we know leads to many disturbances of digestion and bowel function, which means irregular peristalsis in the bowel and appendix.

The degree of inflammation in the appendix must vary greatly as the degree of inflammation varies elsewhere in the body. It may be part of a general process accom-

panying inflammations of other lymphoid tissues, *i.e.*, tonsils and adenoids. It may accompany a general enteritis, clearing up spontaneously with the general condition or it may continue locally after the general infection subsides. If there is any parallel between inflammation in the appendix and inflammation elsewhere in the body or skin, where a very small percentage rarely reach a stage where a doctor is consulted, then the great mass of people with mild appendicitis never see a physician. This is borne out by my own experience in seeing over a period of years the nurses in our training school (almost 300). The doctor is called for even vague symptoms. Early

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symptoms often clear up quickly, though definitely suggestive of appendix disease. We do not advise operation until several hours elapse to see whether symptoms clear up or get worse. Repeated mild or unrecognized attacks undoubtedly cause changes in the appendix similar to those the pathologists call chronic appendicitis.

I have prepared a thermometer-like diagram to illustrate our ideas of inflammatory diseases of the appendix. Zero represents the normal appendix—the appendix at birth or before there has been any irritation. The gangrenous, perforated or pus appendix is represented by 100°. Between the o and 100° there is room for every conceivable grade of inflammation. I venture to say that the surgeon sees only cases where the inflammation has reached well up toward the top of this scale in seriousness. Many of the transient stomach aches and cramps of adults are probably an expression of mild inflammations of the appendix which go undiagnosed. Many pathologists agree with Aschoff that it is the recurrence of such acute attacks that cause the so-called chronic appendix.

Osler also wrote that chronic appendicitis followed acute attacks or it might be chronic from the beginning. During my years on the resident staff at the Johns Hopkins Hospital the diagnosis of "acute exacerbation of chronic appendicitis" was a very common one, and from the present survey I believe that it is a very sound diagnosis to make, even though chronic appendicitis is accepted not as a continuous process but as an abnormal appendix, resulting from repeated mild recurring acute attacks.

Whether a pathologically chronic appendix, however, causes clinical symptoms seems very doubtful. If we take Aschoff's view that there are recurring mild inflammatory attacks, we can sometimes make the diagnosis during a mild inflammation.

The following tables show a study of the pathological material at Henry Ford Hospital from 1923 to October, 1930. The first three show pathological findings in appendices diagnosed appendicitis followed by appendectomy.

TABLE IV	TABLE VI
Acute appendicitis	Chronic appendicitis
Acute diffuse appendicitis	Total chronic cases
Total acute cases 503	Total
TABLE V Subacute appendicitis 58	Normal appendix

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The following tables show pathological findings in appendices removed as a routine during operations on the pelvic organs and gall-bladder.

TABLE VII	TABLE VIII
Operations on uterus alone 716	Tubal pregnancy 57
Appendicitis, chronic	Appendicitis, chronic
Total appendices removed	Total
Appendix removed in 67 per cent, of operated cases	Total No. appendices removed 27 Pathological findings in 93 per cent. Appendix removed in 47.5 per cent. of operated cases
TABLE IX	TABLE X
Salpingitis, chronic, acute and subacute	Disease of the ovary
Appendix removed in 75 per cent. of oper- ated cases	- VI
	E XI
Total operations on wall bladder	7.77

Total operations on gall-bladder	
Appendicitis, chronic	203
Appendicitis, chronic obliterative	114
Appendicitis, acute	4
Appendicitis, subacute	2
Total	
Appendix normal	(
Total appendices removed	419
Pathological findings in 98.6 per cent.	
Appendix removed in 57 per cent. of operated cases	

Table XII sums up pathological findings in appendices removed with diagnosis of appendicitis and during operations on gall-bladder and pelvic organs.

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TABLE XII

Total appendices removed with diagnosis of appendicitis	. 1508
Normal appendices in	13
Pathological findings in	99.04%
Total No. of appendices removed during operations on pelvic	
organs or on gall-bladder	1589
Normal appendices in series	67
Pathological findings in	95.79%
T-tol apponding removed	200
Total appendices removed	3007

Table XII, the summary of all charts, shows that about 95 per cent. of appendices reported back from the laboratory have pathological changes.

A recent survey not yet completed of the case histories of appendicitis operated on since the opening of the Henry Ford Hospital to 1929 only, show that there have been 940 operations for appendicitis with resulting mortality of 6.5 per cent. in acute cases while in the series of 500 interval cases the mortality has been .2 of 1 per cent.* This latter mortality was dependent on acute myocarditis. This means one death in 500 operative cases.

Appendices removed at autopsy show a much smaller percentage of chronic appendicitis than those removed during operation upon the upper abdomen. Kraussold, Deaver and Toft report pathological changes in appendices in about one-third of all autopsies. Heyd⁶ reports the ordinary post-mortem incidence of disease of the appendix to be about 17 per cent. Eusterman⁵ found the appendix diseased in 48 per cent. of gastroduodenal ulcers. Heyd reports disease of appendix in 69 per cent. of cholecystectomies and in 54 per cent. gastric ulcer. Deaver and Ravdin⁴ in seventy-six cases found disease of the appendix in 90 per cent. of gall-bladder disease and 61 per cent. of gastroduodenal ulcer. This is suggestive of the significance of appendicitis as a possible forerunner of upper abdominal trouble such as cholecystitis or ulcer.

We schedule for operation, as interval or potential appendicitis, those cases which give a history of one or more acute attacks. The pathological report comes back, chronic appendicitis, in these cases.

Exploratory incision should always be made when operating for interval appendicitis even though thorough studies may have failed to show disease of biliary, urinary or other gastro-intestinal tracts. Even with negative clinical history and findings there is an occasional surprise at operation.

If we could establish a standard for a clinical diagnosis of the more advanced so-called chronic appendix cases, or perhaps better called recurring

^{*}We have been considerably annoyed by the quotation of statistics published in Jour. Am. Med. Assoc., February, 1926, on "Factors Influencing Appendicitis Mortality" by Dr. F. C. Warnshuis, in which the following figures were given from the Henry Ford Hospital: 6.9 per cent. mortality in acute cases; 3.08 per cent. in chronic cases. These figures are incorrect and do not come from any proper statistics from this hospital.

mild appendicitis, which gives none of the classical signs of appendicitis, we might accomplish something worth while. It seems that an acute attack of appendicitis is prone to leave changes in the appendix which lead to a diagnosis of chronic appendicitis by the pathologist.

It is generally agreed if one has had acute attacks without the operation that the appendix should be removed during the interval between attacks, especially if the individual concerned should be going on a journey where prompt good service is not assured in case of a sudden attack. We all know of tragedies where this has not been done. Definite tenderness elicited by direct pressure over the appendix when visualized by the fluoroscope after barium is significant of so-called chronic appendicitis, or a mild recurring attack. If, however, there is tenderness along the course of the entire colon, these cases are regarded by us as functional ones and treated as such with a diagnosis of irritable colon or neurogenic colon. Dr. John Mateer, our colleague, has convinced us of the existence of such a condition.

That diagnosis of abdominal conditions today is much better than that of twenty-five years ago, there can be no doubt as evidenced by the constantly decreasing numbers of exploratory operations. Today there should be no operations for appendicitis when the pathology is in the biliary or urinary system. The neurogenic or irritable colon, so often a purely functional disturbance, can be ruled out as can pylorospasm by the therapeutic test of rest and a regulated bland diet.

The increasing mortality rate indicates that laymen are sadly in need of being made even more appendix conscious than they are at the present time, though many argue to the contrary.

Discussion.—1. The mortality rate from appendicitis is steadily increasing.

- 2. There is marked sentiment against the difficult clinical diagnosis of chronic appendicitis even though pathologists diagnose as chronic appendicitis, 85 per cent. or more of appendices removed during the course of other operations.
- 3. The finding of a chronically changed appendix in such a large percentage of cases, coëxisting with cholecystitis and ulcers around the pylorus, in view of the close association with the lyphatic drainage from the appendix, is suggestive of a possible etiology of the former condition in certain cases. McCarty finds that the average age of gall-bladder climax is ten to eleven years beyond that of the appendix peak.
- 4. Causes of increased appendicitis rate in this country may be attributable, perhaps, to type of food, bowel habits, nervous tension and worry interfering with normal peristalsis, as well as increased infections of the upper respiratory tract.
- 5. Clinical diagnosis of pathological lesions of the appendix has not kept pace with advances in biliary- and urinary-tract diagnosis.
- 6. The chief bone of contention in the literature seems to be over the fact that the pathologists, who do not all agree, mean by chronic appendicitis chronic changes in the appendix due to inflammation, usually previous,

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whereas the clinicians can find but little clinical signs of it. (This is also often true with root abscesses or sinusitis—except by X-ray.)

7. Criticism of the operation of appendectomy where incomplete diagnostic studies were made before operation is justifiable but such criticism of the operation because symptoms are not relieved, even though a markedly diseased appendix was removed, is not fair. Appendectomy for a definitely diseased appendix in view of almost 18,000 deaths from appendicitis, must have a higher purpose than the relief of symptoms.

8. The majority of patients with attacks of appendicitis showing mild symptoms probably never consult a physician and in many cases where the patient is seen by the physician, the signs are so slight that the diagnosis is not made.

9. The great numbers of operations done in some localities for chronic appendicitis has led to unkind and perhaps just criticism of surgical judgment, and this paper is not meant to be taken as an approval of such wholesale removals of the appendix.

CONCLUSIONS

I believe that so-called chronic appendicitis cannot today be fairly considered, excepting as linked with mild or severe recurring acute inflammations, because

1. The discussion concerning the existence of such a condition has become rather acrimonious—some writers denying its existence with an intolerant wartime spirit.

 The pathologist interprets scarring from repeated inflammation as chronic appendicitis while some clinicians deny any clinical entity of chronic appendicitis.

3. As a result there is a large class of cases with recurring mild appendicitis with insufficient signs to make a diagnosis. These cases are usually overlooked until they flare up into acute fulminating appendicitis.

4. In view of the huge increasing mortality from appendicitis in this country, time now spent in discussions which are as footless as those concerning the number of angels who could stand on a needle point, since neither occupy space, had much better be spent in the study of better and earlier diagnosis of the mild recurring attacks of appendicitis if we are going to make any headway against the rising tide of deaths from appendicitis and its many complications.

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BIBLIOGRAPHY

- ¹ Aschoff, L.: Chronic Appendicitis. Med. Klin., vol. iv, pp. 1660-1661, 1928.
- ² Carr, C. I., and Deacon, W. G. C.: Appendicitis. A Study of Michigan Statistics. Jour. Mich. State Med. Soc., vol. xxvi, pp. 258-262, 1927.
- ³ Deaver, J. B.: Appendicitis, Its Diagnosis and Treatment. Blackiston, 4th edition, 1914.
- Deaver, J. B., and Ravdin, I. S.: End Results in 500 Cases of Appendicitis. Arch. Surg., vol. vi, pp. 31-40, 1923.
- ⁵ Eusterman, G. B.: Essential Factors in the Diagnosis of Chronic Gastric and Duodenal Ulcers. Jour. Am. Med. Assoc., vol. cxv, pp. 1500-1503, 1915.
- ⁶ Heyd, C. G.: Chronic Appendicitis. Surg. Clin. of North Amer., vol. i, pp. 519-533, 1021.
- ⁷ Hoffman, F. L.: The Appendicitis Record for 1928. The Spectator Company, New York.
- ⁶ Public Health Reports, United States Public Health Service, p. 2755, November 7, 1930.
- ⁹ Ryan, T. G.: The Mortality from Appendicitis. Am. Surg., vol. xci, pp. 714-717, 1930.
- ¹⁰ Selinger, J.: Chronic Appendicitis. Surg. Clin. of North Amer., vol. viii, pp. 309-319, 1928.
- ¹¹ Warnshuis, F. C.: Factors Influencing Appendicitis Mortality. Jour. Am. Med. Assoc., vol. lxxxvi, pp. 469-471, 1926.

TRAUMATIC INDUSTRIAL CANCER

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In those states, such as New York, where laws have been enacted protecting the workman against occupational hazards and compensating him for injuries or diseases resulting from such casualties, real importance attaches to the question of whether or not cancer developed because of the nature of the industry per se or from an injury acquired in the course of the work.

Since the cause of cancer continues to be a mystery, it cannot be stated definitely that cancer developing on the site of an injury is or is not due to such injury and whether the injury itself produced the cancer is also still questionable. In the opinion of Connheim, there are at all times embryonic rests in the human being which are started on an outlaw growth by some outside stimulus and form cancer. Virchow, on the other hand, believes that cancer is in the normal blood-stream and suddenly, because of some unexplained extraneous factor, ignoring all the customary rules of normal body tissue, grows in an abnormal manner and forms cancer.

Although, however, the exact cause of cancer is unknown we have learned from experience that persistent irritation serves to stimulate the transformation of normal to abnormal tissue. Whether, for this to occur, a so-called pre-cancerous condition must first exist is likewise still undetermined, for to date no definite description can be given of this indefinable, precancerous stage. Nevertheless, by reason of our observations, we are led to believe that certain conditions do tend toward preparing the body tissues for the development of a malignant growth in some special area.

Thus infection, especially syphilitic, is often likely to be such a preparatory cancer agency. It is our belief that cancer does not develop by any single agency; it takes several factors, combined, to bring on the malignant result, and when once the field is properly prepared, any one contributing factor may be sufficient to shove the normal tissue over the border line into a state of cancer.

The question of interest to us at this time is whether cancer is an industrial disease. Does it occur as a result of industrial hazard, or does cancer follow upon some injury received during the course of one's occupation?

In our cancer work in the Division of Cancer, Department of Hospitals, City of New York, we have had the opportunity of observing some two thousand cases a year in persons ordinarily classified as laborers or industrial workers. To the question how often is the presence of a malignant condition due to industry, examination of our records discloses no industrial factor properly important enough to guide us. Cancer of the stomach has

occurred in office workers, as well as in normal laborers; œsophageal cancers in workers of sedentary habits and in those always doing outside work; in fact, our records show no specially significant industrial factor save in the one instance of X-ray workers, where we have direct evidence of malignant development of an X-ray injury. However, we do have peculiar incriminating circumstances which, if we believe that persistent irritation causes or stimulates the development of cancer, are manifested by some of the lesions appearing in patients presenting themselves to us for treatment.

Taking up first the matter of X-ray cancers: In the case of those technicians or physicians employed in the business of taking X-ray pictures, persistent exposure to the X-rays leads to an intractable burn which, as we have seen in numerous instances, develops into a cancer. Then there is the working man who, due to injury, is required to have frequent radiographic examinations. The area so exposed to the X-rays may later on appear burned and on it may occur a malignancy. It is rather characteristic of this type that injury burns appear very late following exposure to the rays. Shall such a case be classified as an industrial cancer and the patient entitled to compensation relief? Often we see patients who are referred to us for a cancerous growth developing on the skin effected by frequent X-ray exposures in the examination of fractured bones received in an injury in the course of employment. Shall we consider the development of cancer on the site of the X-ray burn a result of industrial trauma? The frequent examinations were given without due consideration of the possible resultant ill effect on the skin, although carried out, of course, with the idea of assisting in the therapy following an industrial accident. The skin so often exposed to the repeated X-rays broke down and on the ulcerated area a malignancy developed. Is the accident then responsible for this cancer development?

Another case is that of the shoemaker who constantly carried tacks in his mouth. Persistent irritation of the mucosa of the roof of the mouth brought on a sore, which following continued irritation by the tacks developed into a malignancy. Is this an industrial cancer?

There is the case of the working man, aged thirty-three years, who finds out he has a malignancy of the upper arm following the not unusual injury of being struck on the arm by a broken engine belt. Up to the time of his injury he worked every day and not until a radiograph of his arm was taken a week after the accident was the presence of a malignant bone condition noted. Was this a quiescent malignancy which was stirred up by the accident? Would it always have remained quiescent except for the accidental injury? Or did he have a growing tumor, insidiously progressing within him, and ready to break forth, so that only the occurrence of the accident called attention to its presence?

Similarly the case of the laborer who, in falling, struck against some protruding object in such manner as to painfully contuse his scrotum and

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testicle. A month later he is confined indoors with fever and bloody expectoration. Examination shows a large tumor mass in the scrotum, swollen inguinal glands, a hard pelvic mass, with radiographs of the chest showing a typical picture of metastatic malignancy. This case rapidly went to exodus and an autopsy proved the presence of a testicle cancer with generalized body metastases. Did this condition arise from the injury received during the course of his work or had the disease existed prior to the accident and was lying dormant only to start its rapid destructive course under the stimulus of the accidental injury?

From experience we know that the ordinary mole lies quiescent in the human body, but when irritated becomes very malignant, rapidly destroying its host. May we blame the death from a generalized melanosarcoma in a carpenter to a saw injury to his finger six months previous? Did the saw, cutting into his finger while working in the shop, stir up this hidden cancer focus which rapidly ended the carpenter's life?

A bricklayer develops a sore on the back of his hand which does not heal because his hand is always in lime mortar. Examination of the sore shows it to be a skin cancer. From what did it develop and is the bricklayer's occupation responsible for the cancer's presence?

Another case is that of a tailor, whose job was sewing garments on a motorized sewing machine. One day the motor belt broke and the free end, striking a wart on his forehead, caused a bleeding wound which persisted for a long time in spite of the usual medications applied to surface wounds. Examination of the tissue of the lesion showed a skin cancer. Why did it develop and is it attributed to his occupation?

Take now the case of the girl, who is burned in a fire in the factory where she works, and is permanently scarred over her body: in places the scar tissue has broken down and a persistent ulcer is annoying. Examination of the bordering tissue shows evidence of the presence of cancer in this scar tissue. How did it occur and what was responsible for its development in this particular spot? Must her employer compensate her for this injury?

Another case recently observed was of a young woman of thirty, ordinarily very healthy, employed as a salesgirl in a large department store. In the course of her work she had to reach for articles on several shelves near by and frequently she struck her breast against a shelf. She paid no attention to these bruises but one day she hurt a breast more severely than usual and reported to the company doctor seeking relief from the pain. He noted merely a slight bruise and a rash, and prescribed a salve. Gradually the breast began to swell and the doctor then noted the presence of glandular enlargement in the axilla. He sent the girl to the hospital for removal of an axillary node for diagnosis. The microscopic picture was that of a carcinoma and radical removal of the breast followed. Four months later the girl succumbed from a generalized carcinomatosis. What caused the cancer? Was it persistent bruising of the breast, or did the injury merely

stimulate an already present malignancy? Was dissemination of the cancer expedited by the injury? Is it a cancer of industrial traumatic origin?

A man working in a repair shop is struck on the heel with a plank. The heel is bruised, later becomes infected and the skin ulcerated. The usual dressings fail to make the lesion heal. A biopsy of the ulcerated area shows the presence of carcinoma. Is it a result of the injury?

Another case of a leg injury was that of a stone mason, admitted to the Cancer Institute in December, 1928, with the history of having been struck on the left thigh with a block of building stone ten weeks before. He was but thirty-eight years of age; examination showed a swelling of the left leg, X-rays showed a malignancy of the bone. Amputation of the leg proved the diagnosis. Did the injury cause the malignancy?

In another case a machinist sustained a blow upon his arm by a flying piece of chipped metal. A large ulcerated tumor develops over the site of the arm which bled profusely, necessitating his stopping work and his admittance to the hospital. Microscopic diagnosis of the tumor tissue shows it to be a sarcoma. Was his injury responsible for this new growth?

A man working in an ice cream factory claims he struck his chest against the machine and from that time on had difficulty in breathing. He was admitted to the Cancer Clinic last summer with the chest full of malignant tissue as shown by the X-ray picture. X-ray therapy caused a large part of this tumor to disappear. On this result we base our diagnosis of lymphosarcoma. A gland removed for biopsy proved the correctness of this diagnosis. The patient, however, succumbed to the disease and the family are now claiming compensation on the ground of traumatic cause for the disease. Are they justified? Is this a traumatic malignancy?

What can be our answer in these cases? Is industry to blame or are these occurrences of cancer mere coincidences? Knox² from her thorough review of the literature of traumatic cancers and her own records definitely states that conclusive proof of trauma as a causative agent is still lacking. While some cases seem to suggest the possibility of cancer due to a single trauma, positive proof in these cases is lacking. She believes that in many instances trauma merely calls attention to an already existing malignancy. Wells and Cannon⁴ reported a case of apparent traumatic cancer of the lung, where the lesion developed immediately following trauma, and autopsy showed tumor development beginning at the seat of the injury.

Mock and Ellis,³ in a study of trauma and malignancy, conclude that there is no justification for assigning trauma as the etiological factor for cancer unless scientific proof is definitely given. Ewing¹ has established certain rules upon which he bases his opinion as to the traumatic origin of a tumor. These are: The authenticity and sufficient severity of the trauma; previous integrity of the wounded part; the identity of the injured area with that giving origin to the tumor; the tumor must be of a type that could conceivably result from trauma, and there must be a proper time interval

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between the receipt of the injury and the appearance of the tumor. One point often is brought up for discussion, what has the general condition of the patient to do with the formation of cancer following trauma? Does the weakening of general body health due to injury to a part of the body accelerate malignant growth? From our experience we feel that malignant growth is not affected by the general state of the body.

From our observations we have noted that there is one factor in all these cases of supposed traumatic malignancy and that is that some sudden injury calls attention to the lesion. Has a long-time, persistent injury been at work, such as we believe is a necessary causative agent to produce cancer, only waiting for the recorded trauma to manifest itself? Was cancer present for a long time previous to injury and only needed the trauma to start it off to rapid development? The time usually given, however, as elapsing between the injury and the malignant manifestations is very short. What then does it? Perhaps some day we will find an answer to all these problems with the discovery of what cancer really is and the cause of its occurrence.

REFERENCES

¹ Ewing, James: American Journal of Surgery, February, 1926.

⁸ Knox, Leila C.: Archives of Pathology, vol. vii, February, 1929.

³ Mock, H. E., and Ellis, J. D.: American Medical Association Journal, vol. lxxxvi, January 23, 1926.

Wells, G., and Cannon, P. R.: Transactions of Chicago Pathological Society, June 1, 1930.

FOREIGN BODIES IN THE STOMACH AND INTESTINES*

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Foreign bodies in the stomach and intestines come under two general classifications, those that have been formed in the stomach or intestines (bezoars) and those swallowed intentionally or otherwise.

Bezoars are bodies which are formed in the stomach and intestines. They are of various types, such as trychobezoars, phytobezoars, etc. In some cases the bezoar results from certain articles of food or drink.

The word bezoar comes from the Arabic bazor, bedsoar, bezahar or badizorh, Persian padzorh, or Hebrew bel or baal zaar-that which masters or counteracts poison, or an antidote. Reduced to a two-syllable word later; is apparently of modern Latin origin. Later the expression bezoar stone is noted. Until the eighteenth century writings on pharmacy mention Lapis Bezoar and state that it is usually obtained from the stomach or intestines of the Persian wild goat; sometimes found in certain variety of antelope. This concretion was the source of the tincture, also pulvis bezoardicus. They were highly prized and were used as remedies against pestilence as well as poison. They were also worn as charms. Galen, 130 to 200 A.D., mentions bezar in connection with the treatment of the bite of a rabid dog and states that it may save the patient's life. Certainly from the tenth up until the eighteenth century they were held in great esteem by the medical profession. Avicenna, the Ibn Sina (A.D. 980 to 1037) of Arabian medicine, says in his "Canon of Medicine," "Communes medicinæ ad venena al bezahar," mentioning theriac and cardamon also. Ambrose Paré, 1517-1590, says of bezoar that it was used by the Arabs and Persians and was also known to the Greeks. He mentions oriental and occidental bezoars (the latter from the stomach of the llama in South America), also describes the usual tests for determining whether true or false. However, Paré was dubious about their efficacy and states that with the cooperation of Charles IX he conducted an experiment on a condemned criminal by giving him bichloride of mercury in order to prove the worthlessness of bezoar as an antidote for poison. Wilhelm Fabry of Hilden, better known as Fabricius Hildanus, 1560-1624, usually called the Father of German Surgery, refers to the medicinal use of the bezoar times without number. He particularly recommends it as a remedy for dysentery and states that twenty grains is the usual adult dose. Benzoar was apparently often used in conjunction with the universal antidotes of the period, mithridatium and theriac or theriaca.

Michael Ettmuller, 1644-1683, was a member of the faculty of Medicine at Leipsic in 1676. About this time he was appointed professor of botany,

^{*} Read before the Southern Surgical Association, December 11, 1930.

also of surgery and anatomy. He enjoyed a great reputation as a teacher and wrote extensively. An edition of his writings published at Leyden in 1690, contains the following: "Qui tamen bezoar in peste et morbis malignis potentissimi dictus medicina." About this time Van Helmont, 1577–1644, of Brabant, was recommending bezoar in the treatment of pest, malignant fever and stone; Lieutaud used it for variola, Valentinus likewise for the relief of epilepsy. Blanton tells us that the physicians of early colonial Virginia used bezoar as a remedy for snakebite.

The old idea of the madstone is obviously a hangover from the empirical age of medicine when bezoar was a highly prized remedy in general use. Even at this time, we occasionally hear of the madstone being used, especially for the prevention of rabies. Bezoars, or large concretions, are found at times in the stomach or intestines of various ruminant animals; this type of stone has in many instances apparently been built up in layers. Some of the specimens seen in the Museum of the Royal College of Surgeons in London, England, are in shape not unlike the stone pestles used by the American Indians for pounding and grinding corn.

One of the most extensive studies of hairballs or trychobezoars has been made by Matas. He mentions the first case of this kind reported by M. Baudamant in the *Journal de Médecine*, Paris, 1779. He calls attention to the esteem in which bezoars were held by the ancient Hindoos and Persians as evidenced by the writings of Susruta in the fourteenth century before Christ and by the manuscript on drugs written by Muwaffak in the tenth century A.D. He states that the hair-swallowing habit is most often observed in young girls. In one case the hairball was formed of bristles. The patient, a woman, was a brushmaker. In some cases the mass may be made up partly of hair and partly of other foreign material.

Hairballs apparently may be present for long periods of time without causing symptoms, probably due to their softness and their adaptability to the normal contour of the stomach. Generally, however, foreign material in the stomach causes thickening of the stomach wall and even ulceration, rather rarely perforation. Hairballs have several times been found that weighed as much as $2\frac{1}{2}$ to 3 pounds. However, Swain in 1895 reported one that weighed 5 pounds and 3 ounces. While most of the cases recorded have occurred in women, Hart mentions a case of hairball occurring in a man who was in the habit of chewing his long beard.

Children even form such habits as hair-swallowing. Doolin published a clinical note concerning a case of hairball of the stomach removed from a little boy, three years of age. The patient began to complain of pain in his stomach one week previously and vomited from time to time. He was unable to obtain any clinical history that would give any idea as to the source of the mass. The child subsequently proved himself to be a boy of normal intelligence and good disposition.

Veterinary surgeons state that it is not uncommon for them to find hairballs in the stomach and intestines of cats, particularly of the long-haired

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Persian or Angora types. A local veterinary has removed several hairballs from one cat on different occasions.

With reference to the formation of bezoars in the stomach of the human being, it is thought that the mass is formed at one time and that it may be due to the rapid satisfaction of a ravenous appetite by some material that is plastic and not readily absorbable.

These masses usually remain in the stomach but have been known to pass through a large gastroenterostomy opening and to lodge in the small bowel.

Porter, Balfour, Peple, Hart and Outten report notable examples of phytobezoar resulting from the eating of persimmons. Apparently it is not always necessary to eat large quantities of the fruit; however, the usual history is that the individual was hungry and that large amounts of persimmon were eaten.

Prasad found a mass in the stomach of a twenty-five-year-old laborer who had been under his observation for six months. The tumor caused uneasiness after a full meal. When removed it was found to be $6\frac{1}{2}$ inches long by $2\frac{1}{2}$ inches broad and composed of a dense, dark-green substance found later to be caoutchouc. The patient remembered having taken the latex, or inner bark of the banyan tree (Ficus Indica), mixed with sugar, as a tonic. This was in keeping with the findings because the bark of the banyan tree contains caoutchouc which is not affected by the gastric juice.

Kortmann and others reported shellac stones, weighing as much as 2,000 grams, removed from the stomachs of individuals who worked in porcelain factories and drank alcoholic solutions containing shellac. When this comes in contact with the watery contents of the stomach it becomes solidified.

A bezoar formed from pumpkin residue as a basis was removed from the stomach of a Chinese by Adams. There had been persistent vomiting, loss of weight and gastric discomfort for five or six months. In view of the enormous mass in the stomach, a diagnosis had been made of advanced carcinoma. Surgical exploration was done at the urgent request of the patient.

Hamdi reported a bezoar made up of salol taken from the stomach of a Turk who had taken large doses of salol over a long period of time for the relief of cystitis. He also reported three cases of bezoar removed from the stomachs of men who had, under the stress of war, lived from two to four weeks in the open fields, eating grass, plant roots, etc.

Kummant likewise reports a bezoar made up of bismuth which had been taken for X-ray study of the gastro-intestinal tract.

Bucknill removed a phytobezoar made up of string and cocoanut fibre which had been swallowed by an insane patient. Schreiber found one in the stomach of a patient who habitually ate salsify.

Martin reported a case in which a bezoar, thought to consist almost entirely of tobacco, was removed from the stomach of a tobacco chewer.

My associate in surgery, Dr. Bankhead Banks, observed a foodball made up of agglutinated watermelon seeds in the large bowel of a Negro man who gave a history of having eaten four or five watermelons in the course of one afternoon. This was broken up and passed naturally.

It is difficult and almost impossible to arrive at the incident of tricho- and phytobezoar. A few observers have reported more than one case. Hart, however, has reported eight cases that may be associated with this condition. Reviewing the literature, Limbaugh reported two cases and abstracted 108. He observed that the hairball more frequently occurs in persons of normal mentality than in the insane.

Rivers and Davison cite three cases of persimmon bezoar and one of rags and twine. They call attention to the fact that gall-stones at times ulcerate into the stomach or intestines where they may cause the usual symptoms incident to foreign bodies. They

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mention the following symptoms that have been observed as a result of the intravisceral presence of such bodies: General symptoms, such as collapse, diarrhœa, poor appetite, convulsions in children; symptoms secondary to the presence of large foreign bodies, such as weakness, loss of weight and anæmia; disturbances of gastric motility, as flatulence, nausea, regurgitation or delayed emptying time.

Obviously in a number of cases pre-operative diagnosis by X-ray has been made. The X-ray diagnosis based on the barium meal gives a most distinctive picture. The mass is shown in outline surrounded by a film of barium. Under the fluoroscope the mass can be moved about in the stomach. Matas states that the liquid barium spreads out and surrounds something in the intragastric mass. The diagnosis is based on a mass in the stomach. When the barium meal is given, the stream spreads over the object and the shadow under the fluoroscope looks like an empty space which is freely movable. After the meal has passed into the intestine the barium may be seen to form a cap in the stomach between the mass and the stomach walls. This mass may be displaced upward and sidewise by manipulation.

A few cases have been cited by King in which the bezoar was broken up by massage. In most of the cases the ball has been removed by gastrotomy; in a few enterotomy also had to be performed; a few were removed by enterotomy and in a few both gastrotomy and enterotomy had to be performed.

Foreign bodies are swallowed either by accident or by design, by the insane and mentally unbalanced and by jugglers or mountebanks for professional gain. At times the swallowing of foreign bodies is doubtless done with suicidal intent. However, in many cases it is apparently due to a strange perversion of the normal appetite. An unusual case was recently reported in the public press: A small boy and his companion agreed to see who could swallow nails the faster. The guileful one pretended to swallow the metal and did not while the honest one swallowed a large quantity of nails and died. In another instance a little girl watched her father, who was a lather, putting nails in his mouth. She thought he was eating them, tried to imitate him and died as a result.

Some years ago a patient on whom I had operated for the closure of a recently perforated duodenal ulcer, told me that for more than a year, acting on the advice of a native healer, he had daily swallowed a teaspoonful of river sand and fully believed that his ulcer was being cured by this means.

Many of these patients say that they swallow these articles in response to an irresistible impulse, as witness the following spectacular news item concerning a patient of one Doctor Fuchs in a State Hospital for the Insane, Munich, Germany. "Since he had swallowed needles, nails, knifeblades, spoons, a screwdriver handle, a beer seidel handle, coins, matches, etc., to such an extent that he was allowed no clothing with buttons, he readily discovered a new source of false foods—his fellow inmates. He would pounce upon an unwary victim, trip him up and, snarling, chew off every button, every fastening from his clothing and swallow the same."

When these bodies are small and not swallowed in excessive numbers

they usually pass through the stomach and intestines and, as a general rule, without lodging anywhere for an undue length of time or causing any serious trouble. However, if large numbers are swallowed at one time, or for some reason accumulate, they become interlocked together in such a way that it is relatively impossible for any of the articles to break away and pass on.

Sharp objects such as needles, pins, knifeblades, etc., often run the gamut of various intestinal kinks and peristaltic waves without apparent injury. One of the most striking cases reported is that of Allison who observed a patient who had swallowed a piece of razor blade. This traversed the intestinal tract and was evacuated within forty-eight hours. Kern reported a case in which a piece of fork, 14 centimetres long, passed through the bowel naturally.

At times a foreign body is passed after having remained *in situ* as it were for months or even years, as witness the case reported by Smith of a woman who passed a darning needle twenty years after she had swallowed it.

Foreign bodies tend to lodge and remain in two distinct locations: First, the stomach which ultimately as a result of the continuous weight sags down into the pelvis and tends to keep the foreign bodies in one position. Secondly, these masses frequently accumulate in the dependent cæcum. Here again gravity plays its part. In this location they sometimes cause perforations. Several such cases have been reported. MacEwen's case had peritonitis from perforation of the cæcal wall by a straight pin. Pike reported a perforation of the cæcum by a three-inch wire nail. Non-metallic objects infrequently cause perforation; however, Ginsburg and Beller reported fifteen cases of this type of surgical complication due to fish- and chicken bones and toothpicks.

Myerson has called attention to the fact that professional swallowers take into their stomachs numerous articles of various sorts and as a rule pass them by rectum. They come to grief, however, when the collection lodges in the stomach or cæcum and forms a definitely obstructing mass. He stated that in a large majority of the instances of surgical complications arising from foreign bodies, there has been no definite obstruction. He further says that in a large majority of the instances of surgical complications arising from the swallowing of foreign bodies, there has been no definite history of the ingestion of the foreign body.

The rectum is usually most tolerant to foreign bodies and they here cause but little trouble beyond the possible ulceration, incident to prolonged pressure in one location.

Foreign bodies are not infrequently swallowed by accident, as was the case of a table knife swallowed by the professional juggler, Hermann, reported by Bernays. The juggler was holding the end of the knife handle far back in his jaws between the wisdom teeth and let it get away from him. Pins are often swallowed accidentally by infants and small children.

Little reports perforation of the stomach by a lead pencil which had been swallowed some time previously. When found at operation the graphite had dissolved out of the pencil.

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Lupton cites a case of inadvertent swallowing of a long piece of wire which perforated the stomach. However, Allison describes a case in which a long piece of wire passed through the alimentary canal without causing apparent injury.

A review of the literature shows that perforations occasionally occur from wooden toothpicks, fishbones, nails and other sharp objects. Large quantities of nails and other hardware that become enmeshed in the stomach tend to remain in that viscus indefinitely.

Rivers and Davison report five metallic foreign-body cases. They mention the fact that instruments, sponges or other articles may be inadvertently left in the hollow viscera during the course of certain abdominal operations. In one such case a whalebone staff and spiral tip which had become separated from a sound being used to dilate an esophageal stricture and lost in the stomach was recovered. They observe that when copper or lead objects have been swallowed, enough of the metal may be absorbed to cause definite symptoms of metallic poisoning.

Vestal, who reported a toothpick perforation of the cæcum, says that the first recorded account of a diseased appendix (Mestiver) presented a foreign body, a large pin, in the appendix.

Experiments made by Exner in regard to giving animals needles and pins found that practically all of the pins and needles passed through the tract without lodging and practically 75 per cent. passed through head-first.

Faber, in his study of the effects of ingestion of fishbones, found that when fishbones are fed to individuals having normal stomach secretions they do not appear in the stools. It is rather remarkable that sharp pieces of bone do not more frequently cause perforation than they do. However, bone remaining in the stomach for any length of time is dissolved by the acid presented in the stomach secretions. This has been observed in numerous instances in which the bone handles of knives have been entirely dissolved in the stomach.

The stomach is apparently most tolerant to foreign bodies. Total weights up to two and one-half pounds, including various small objects by the hundred or thousand in number, have been removed by several different surgeons. These in some instances at least have remained in the stomach for weeks or even months without causing a great deal of disturbance. For instance, Amerson removed a table knife which had remained in a woman's stomach for more than eight months during which time she did her housework.

Fowler found in search of the literature on appendicitis due to foreign bodies, sixtythree cases, but in only five of these was there a definite history of the body's having been swallowed.

Finney, in his paper on the "Development of Stomach Surgery," states that the first reference to a definitely planned stomach operation that he has been able to find was the case reported by Crollius and quoted in Gunther's work on surgery, of a peasant who practised jugglery and was in the habit of concealing a horn-handled knife in his throat. In the course of this manneuvre the knife slipped into his stomach and remained there seven weeks. After plasters and other means of treatment had been employed (among them a magnet in the stomach region), the point of the knife began to cut its way through the abdominal wall. Mathis made an incision and extracted the knife. The date of this operation was 1602. Doctor Finney also quotes from the Memoirs of the Royal Academy, 1743, the removal of a knife from the stomach of a woman who had swallowed it accidentally.

Schwabe, in 1635, is said to have removed a table knife from the stomach of a man forty-one days after its ingestion. Lakin, in 1642, published a similar account of the removal of a "Prussian swallow sword" from a juggler, with recovery.

Baron Larrey in his memoirs, under the subject of "Napoleonic Campaign in Poland," states that he visited Königsberg after the fall of that city in July, 1807. He describes a visit to the town museum and says: "We also saw a small knife which a farmer, named Andreas Guenheid, of Ancient Prussia, had swallowed in 1613. The

violent symptoms which ensued induced Doctor Gruger, a Polish surgeon, to perform gastrotomy upon him; it was done on the 29th of May of the above year, and he lived afterwards ten years." Larrey states further that he remembers while a pupil in the clinic of Frizac, professor of the college of Toulouse, that he saw him operate on a porter, making an incision in the epigastric region, parallel with the linea alba, through which he removed a knifeblade, the point of which had perforated the coats of the stomach. He says that the stomach wound was sutured as well as the integuments and that the wound healed promptly.

Warbasse, in his excellent report published in the Annals of Surgery in 1904, cites a case in which gastric tetany resulted from the mechanical irritation caused by the presence of a large number of foreign bodies in the stomach. The patient underwent a successful operation for the removal of the following articles: Seven pocket knives, seven door keys, twenty large nails, one small spoon, one button hook, one ordinary pin, one knife spring and two watch chains; the total weight being sixteen ounces. He reviews the writings of Mayo Robson, who first presented the surgery of gastric tetany, as regards auto intoxication from gastric fermentation as a predisposing cause, the exciting cause apparently being the reflex irritation caused by painful irritation of the stomach. He quotes Albu, Germain-See and Berlizheimer as having recognized the importance of mechanical gastric irritation as an etiological factor and states that Blazicek has reported a case in which the associated dilatation of the stomach was due to the presence of a large gall stone.

The general symptoms produced by the various foreign bodies in the stomach and intestines of course are similar in character, such as a sense of weight, interference with appetite, pain, nausea and vomiting. There is usually a history of loss of weight.

In the region of the cœcum, perforation has not infrequently taken place and has led to the diagnosis of acute appendicitis, as mentioned by Fowler.

Jackson and Spencer reported two interlocked closed safety pins in the stomach for seven weeks, removed by means of the gastroscope.

Clerf reported the removal of two radium capsules and a rubber finger cot from the stomach and described their removal by the use of the gastroscope, this method having the advantage of being performed without anæsthesia or laparotomy.

As to the methods of removal, they are many and varied. Some of the single not-too-large objects have been removed from the stomach by the use of the œsophagoscope and long forceps.

An ingenious method of removing a safety pin from the stomach was devised by Monteith, who made an abdominal incision and passed a catheter into the stomach through the esophagus. He then palpated the open safety pin through the stomach wall, passed the point of the pin through the end of the catheter and closed the pin. The catheter was withdrawn with the pin intact.

Pratt reports several operations in which straight pins in the cæcum were manipulated into the lumen of the appendix followed by appendectomy.

A single foreign body may be held in the stomach by a narrow pyloric opening. In one case at least, reported by Scott, the surgeon was able under fluoroscopic examination to manipulate a key, turn the handle around and engage it in the pylorus so that the object passed safely *per vias naturales*.

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Collins removed a safety pin from the stomach of a small child by the use of a long forceps passed through a stomach tube, locating the pin by the aid of the fluoroscope. Obviously this method, like the removal of small shell fragments from the lung with forceps and the fluoroscope, the technic of which was so wonderfully perfected during the World War by Petit de la Villeon in Paris, will never become a practical means for the general surgeon.

Usually, however, a large mass of metal objects is meshed together in such a way that a surgical incision into the stomach or intestines is the only method by which they can be removed.

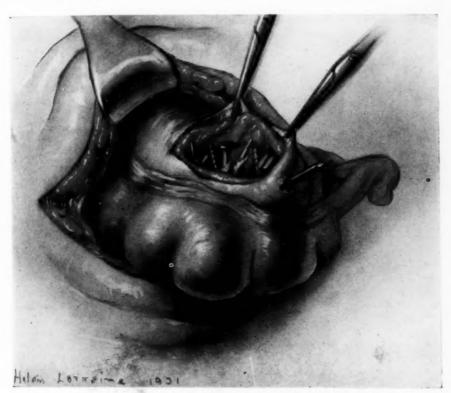


Fig. 1.—Impaction of foreign bodies in cæcum. Perforation by nail,

The literature concerning foreign bodies in the stomach and intestines is voluminous.

Among others, in addition to the cases already cited, notable cases have been reported by Rosenstein who removed from the cæcum two iron plates 15 centimetres in length.

Wolfler and Leiblein published a large monograph on the subject of foreign bodies in the gastro-intestinal tract. The authors observed that the passage of foreign bodies in the gastro-intestinal tract was usually interrupted in the pylorus, the duodenum, the ileocæcal valve and the rectum.

Griffith removed 1½ pounds of metal objects from the stomach of a woman. Chalk reported a case of foreign bodies in the stomach, in 1828, in which he removed more than 2,500 metal objects at operation.

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Thorek reported the case of a foreign-body swallower from whose stomach he removed 276 metal articles besides a great many small pieces of glass and porcelain. He reviewed the literature up to 1924.

Vandivert and Mills, at autopsy, removed more than four pounds of metal articles from the stomach of an insane patient.

Winslow removed nearly 1,300 small objects, mostly straight pins, from the stomach of an insane woman.

Wardell had a patient sixteen years old from whose stomach he removed 21/4 pounds of straight one and one-quarter-inch nails. These had been swallowed over a period of nine years.



Fig. 2.—Mass of foreign bodies, the weight of which dragged the stomach down into the pelvis.

Eliason removed a gall-bladder containing 213 gall-stones and three pounds of metal objects from the stomach of a male patient with recovery.

In 1884, Gussenbauer removed part of a sword, 27 centimetres in length, from the stomach of a sword swallower. The point had penetrated the stomach wall, causing peritonitis, and the patient died.

Halstead, in 1900, removed 74 grams of broken glass and 208 metal articles from the stomach of a juggler.

The following case of recent occurrence contains elements of special dramatic character:

A white female, age twenty-six, single, was admitted to the Charleston General

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Hospital, May 1, 1928, complaining of pain in the right lower quadrant of the abdomen, which was stated to have begun three days previous to her admission to the hospital; some nausea but no vomiting. She acknowledged some indigestion for the past three weeks with marked nausea during the last three days. No gas formation before or after meals. No abdominal pains previous to the present illness. Has lost ten pounds in weight during the past month. Bowels move daily without cathartic.

There was marked rigidity of the right rectus abdominis muscle with marked tenderness in the right lower quadrant. There is a questionable mass palpable in the right lower quadrant. Pelvis not examined.

Under a diagnosis of acute appendicitis she was operated upon shortly after admission to the hospital. On exploring the abdomen, a ten-penny wire nail was found protruding through the cæcum and more hardware could be palpated inside the cæcum. After thoroughly packing off the surrounding viscera, the cæcum was opened and six large wire nails, three two-inch screws, three tacks, one safety pin and one straight pin were removed (Fig. 1).

There was a small amount of localized peritonitis which was fairly well protected by adherent omentum. The incision in the cæcum was closed with tanned catgut and protected with a patch of omentum. No other metallic articles could be found in the

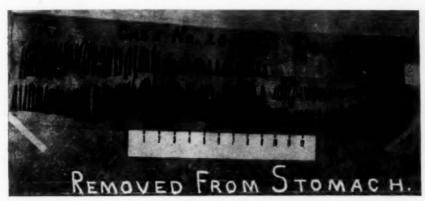


Fig. 3.—First series of foreign bodies removed from stomach.

gastro-intestinal tract. The patient had an uneventful post-operative recovery and left the hospital thirteen days after operation. The wound was entirely healed.

December 10, 1928, a little more than seven months after the first operation, the patient was again admitted to the hospital, this time complaining of nausea, anorexia and vomiting after meals, with history of gradually losing weight for the past two months. She acknowledged having swallowed some nails and a large hard mass was palpated in her lower abdomen on a level with the iliac crest. A radiograph (Fig. 2), of the abdomen showed the stomach filled with metal articles, making a mass 4 inches in diameter. She was operated upon the following morning under ethylene-oxygen anæsthesia. Where the abdomen was entered the stomach was found lying below the crest of the ilium where it had been pulled by the weight of the mass of hardware. An incision 2½ inches long was made in the stomach and the foreign bodies removed carefully, a total of 194 pieces being removed (Fig. 3). A list of them is as follows:

- 105 Wire nails (ranging from six-penny to ten-penny in size)
- 22 Wood screws (some of them as much as four inches in length)
- 18 Finishing nails

5 Stove bolts

15 Roofing nails

3 Safety pins

14 Straight pins

2 Metal book clips

10 Wire hairpins

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The stomach wall, especially the lower portion of the stomach, was markedly hypertrophied and the mucosa was raw and bleeding from irritation. No foreign bodies had passed the pylorus unless they had previously been eliminated. The stomach was sutured with tanned catgut and the suture line protected with gastro-colic omentum.

The patient made an uneventful recovery and left the hospital on the fourteenth day after operation. However, after leaving the hospital she was seen and studied by a neurologist who made a diagnosis of psychopathic personality. She was taken in hand by this neurologist and occupational therapy was started. Rapid improvement was noted after the second operation.

During the month of June, 1929, she again began complaining of anorexia and nausea, and it was noticeable that she was rapidly losing weight. This was just seven

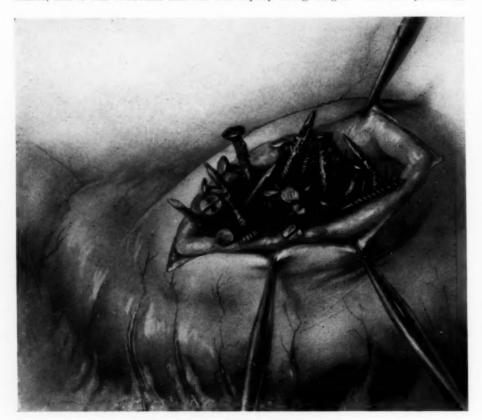


Fig. 4.-Foreign bodies in stomach in situ.

months after the second operation and fourteen months after she was first admitted to the hospital with the mistaken diagnosis of acute appendicitis. At this time, she was still attending to her work and did so until her third operation, on July 1, 1929. At first she would not consent to an examination but after several confidential talks she acknowledged swallowing more nails and permitted a fluoroscopic examination. This revealed a stomach filled with hardware similar to that present at the time of the second operation. She was again operated under ethylene-oxygen anæsthesia, the stomach opened and 140 foreign bodies removed (Figs. 4 and 5), listed as follows:

- 98 Wire nails (ranging from four-penny finishing nails to ten-penny regular wire nails)

 3 Hair pins
- 37 Large wood screws

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It is interesting to note that no evidence of the gastrotomy seven months previously existed at the time of this operation, not even the suture line being visible. The patient made an uneventful recovery and left the hospital eighteen days after operation, the wound entirely healed.

This woman is again going about her social activities, and a special effort is being made to cause her to take more interest in outdoor sports and especially in developing the companionship of young men, both of which she has always shunned. She is now dancing, swimming, taking part in other recreations and enjoying it. Her health has improved and we hope that the vicious circle has been broken for a time at least.

SUMMARY

1. Foreign bodies in the stomach or intestines include bezoars which are formed in situ, and those swallowed intentionally or accidentally. Bezoars



Fig. 5.- Second series of foreign bodies removed from stomach.

are formed from a number of different substances. As basis they may be formed from hair, string fibre, persimmon, pumpkin, raisin, prune, celery, caoutchouc, salol, bismuth and various vegetable materials. The individuals who swallow objects intentionally are the insane or mentally unbalanced, including those who swallow articles with suicidal intent, also mountebanks, or professional jugglers, who eat glass and swallow metallic or other objects as a means of livelihood.

- 2. The symptoms of foreign bodies in the stomach and intestines are, as a rule, those of digestive disturbances due to disturbances of normal gastric motility.
 - 3. The diagnosis, as a general rule, is based on X-ray examination.
- 4. Sharp-pointed foreign bodies result in perforation and its various complications.

- 5. Foreign bodies in the stomach may be vomited; may be passed by bowel, may be removed with the aid of the fluoroscope, gastroscope, but usually by gastrotomy or enterotomy.
- 6. While there have been several reported cases of perforation of the cæcum by wooden toothpicks, perforation of that viscus by nails or other pointed metal objects is comparatively a rare occurrence.
- 7. In the case reported, there were three successive operations for the removal of foreign bodies from either the stomach or intestines and in one instance a perforation had taken place. There has been apparent cessation of the habit in this individual.

BIBLIOGRAPHY

- ¹Galen: Operum Hippocratis Coi Galeni, vol. x, p. 560, Paris, 1679.
- ^a Avicenna: Arabus Medicorum Principis. Version of Gerard of Cremona, Venice, 1608.
- ⁸ Paré, Ambroise: Les Œuvres. Sixième edition, Paris, 1607.
- ⁴ Packard, Francis R.: Ambroise Paré: Life and Times of. New York, 1926.
- ⁸ Fabry, Wilhelm (Hildanus): Medico-Chirurgi Ordinarrii. Frankfort, 1646.
- ⁶ Ettmulleri, Michaelis: Operum Omnium Medico-Phisicorum. Leyden, 1690.
- ⁷ Sennert, Daniel: Opera Omnia, vol. i, p. 56, Leyden, 1676.
- ⁸ Van Helmont, John Baptista: Opera Omnia, p. 144, 1707.
- Lieutaud, Joseph: Synopsis Universæ Praxeos-Medicæ, p. 587, Amsterdam, 1765.
- ¹⁰ Valetinus, Michael Bernhardus: Work on Medicine, p. 300, Frankfort, 1723; also p. 144, Royenburg, 1707.
- ¹¹ Blanton, Wyndham B.: Oriental Bezoar for Snakebite. Medicine in Virginia in the Seventeenth Century, p. 173.
- ¹² Matas, R.: Hairballs or Hair Casts, of the Stomach and Gastro-Intestinal Tract. Surgery, Gynec. and Obstetrics, vol. xxl, pp. 594-608, Chicago, 1915.
- Doolin, W.: A Case of Hairball of the Stomach (Trichobezoar). Irish J. M. Sc., pp. 641-643, Dublin, 1926.
- ¹⁴ Porter, W. B.: Phytobezoar diospyri virginianæ, with report of a case. M. Presse, vol. cxxiii, pp. 175-177, London, 1927.
- ¹⁵ Balfour, D. C.: Phytobezoar Associated with Gastric Ulcer; Especial Reference to Persimmon Bezoar with Report of a Case. Am. J. Surg., vol. vi, pp. 579-587, New York, 1929.
- Peple, W. L.: Globus diospyri virginianae seminum; report of a case. Virginia M. Month., vol. xlviii, pp. 596-599, 1921-1922.
- ¹⁷ Hart, W. E.: Phytobezoars. J. Am. Med. Association, vol. 1xxxi, pp. 1870-1875, Chicago, 1923.
- ¹⁸ Prasad, J.: Raw Caoutchouc in Stomach. Indian M. Gaz., vol. lxii, p. 516, Calcutta, 1927.
- 19 Kortmann: Deutsch Med. Wchnschr., E. No. 26, 1897.
- ³⁹ Adams, W.: Two Cases of Foreign Bodies in Gastro-Intestinal Tract. British J. Surg., vol. xiii, p. 189, London, 1925.
- ²¹ Hamdi, H.: Deutsch Med. Wchnschr., vol. ii, pp. 2122-2123, 1926.
- ²⁸ Kummant, A.: Zentralbl. F. Chir., vol. xlix, pp. 1619–1621, 1922.
- 25 Bucknill: Tr. Path. Soc., vol. v, pp. 45-46, London, 1853-1854.
- ²⁴ Martin, Charles: Personal communication to W. E. Hart.
- ²⁶ Limbaugh, L.: Hairball with Case Report. South. Med. Jour., vol. xxii, pp. 212-214, Birmingham, 1929.
- ²⁸ King, H. P.: Cited by Outten, W. B.: M. Fortnightly, vol. vi, p. 445, 1894.
- Outten, W. B.: A Case of Double Gastrolith Removed by Gastrotomy; Recovery; Death by Phthisis Three Months Afterward. M. Fortnightly, vol. vi, p. 445, 1894.

FOREIGN BODIES IN STOMACH AND INTESTINES

- 28 Rivers, A. B., and Davison, H. L.: Foreign Bodies in the Stomach. Annals of Internal Medicine, vol. iv, p. 742, January, 1931.
- Allison, S. F.: Lancet, vol. ccxiii, p. 968, London, November 5, 1927.
- 8) Kern: Deut. Med. Wchnschr., vol. xlviii, p. 1388, 1922.
- ⁵⁰ Smith, H.: A Needle in the Alimentary Canal. Brit. Med. Jour., vol. i, p. 81, January 17, 1920.
- ³² MacEwen, J. A. C.: Perforation of the Cecum by a Pin. Lancet, vol. ii, p. 785, November 1, 1919.
- ²⁶ Pike, J. B.: Foreign Body in Cecum Simulating Appendicitis. Brit. Med. Jour., vol. ii, p. 413, September 27, 1919.
- ⁵⁴ Ginsburg, L., and Beller, A. J.: The Clinical Manifestations of Non-metallic Perforating Intestinal Foreign Bodies. Annals of Surgery, vol. lxxxvi, pp. 928–940, December, 1927.
- Myerson, M. C.: Foreign Bodies in Stomach and Intestines. Long Island Med. J., vol. xxiii, pp. 410-415, 1929.
- ³⁰ Bernays: Related by Dr. Irwin C. Carlisle, Charleston, W. Va. Personal communication.
- ⁸⁷ Little, Y. A.: Wall of the Stomach Perforated by a Pencil. Jour. A. M. A., vol. lxii, p. 929, Chicago, 1914.
- ³⁹ Lupton, W. J. E.: Another Case of Foreign Body Inadvertently Swallowed. Lancet, vol. ii, pp. 1179–80, London, 1927.
- ²³ Vestal, Paul W.: Perforation of Cecum by Foreign Bodies with Report of a Case Simulating Appendicitis. New England Jour. of Med., vol. cciii, No. 24, p. 1199, December 11, 1930.
- ⁴⁰ Exner, A.: Wie Schuetzi sich der verdauungstract vor verletzungen durch spitze fremdkoerper. Arch. f. d. ges. physiol., vol. lxxxix, pp. 253-280, 1902.
- ⁴¹ Faber, Knud: Ueber graeten als fremkderper im darm und ueber knochenverdauung. Berlin Klin. Woch., vol. xxxv, pp. 768-773, August 29, 1898.
- 48 Amerson: Illinois Med. Jour., vol. xxix, p. 335, 1916.
- ⁴³ Fowler, R. H.: Foreign Body Appendicitis. Annals of Surgery, vol. Ivi, pp. 427-436, 1012.
- "Finney, John M. T.: The Development of Surgery of the Stomach. Annals of Surgery, vol. lxxxi, No. 1, January, 1925.
- Mestiver: Cited by Vestal. A Case of Tumor Situated Near the Umbilical Region on the Right Side Caused by a Large Pin Found in the Vermiform Appendix of the Cecum. Jour. de méd., chir., pharm., vol. x, p. 441, Paris, 1759.
- 18 Schwabe: Berl. Klin. Wchnschr., vol. xx, p. 106, 1883.
- ⁴⁷ Lakin: "A Miraculous Cure of the Prussian Swallow Knife." London, 1642.
- ⁴⁸ Baron Larrey, D. J.: Memoirs. First American Edition, vol. ii, p. 173, Baltimore, 1814.
- ⁴⁹ Jackson, C., and Spencer, W. H.: Safety Pins in the Stomach; Peroral Gastroscopic Removal without Anesthesia. Jour. A. M. A., vol. lxxvi, Chicago, 1921.
- ⁵⁰ Clerf, L. H.: Radium Capsules in Stomach; Gastroscopic Removal. Am. J. Roent. and Rad. Ther., vol. xvii, pp. 635-636, 1927.
- Monteith, W. B. R.: Removal of Foreign Body from Stomach of Infant. Brit. Med. Jour., vol. i, p. 259, London, 1928.
- ⁵³ Pratt, L.: Extraction Through Appendix of Pin in Cecum. Jour. de chir., vol. xv, p. 624, February, 1920.
- Scott, S. C.: Method of Dealing with Swallowed Object. British Med. Jour., vol. i, p. 133, London, 1928.
- ⁵⁴ Collins, C. E.: New Technic for Removal of Open Safety Pin from Stomach. Texas State Jour. Mcd., vol. xxiv, pp. 686-687, Fort Worth, 1929.
- ⁵⁵ Rosenstein, P.: Foreign Bodies in Cecum. Deutsche Med. Wchnschr., vol. xlvii, p. 1099, September 15, 1921.

- Wolfler and Leiblein: Deutsch. Chir., Lfrg. 466, p. 167, 1909.
- ⁶⁷ Griffith, J. D.: Foreign Bodies in Stomach. Jour. A. M. A., vol. lxxxii, p. 31, Chicago, 1924.
- ⁶⁸ Chalk, S. G.: Foreign Bodies in Stomach; Report of Case in Which More Than 2500 Foreign Bodies Were Found. Arch. Surg., vol. xvi, pp. 494-500, Chicago, 1928.
- ⁶⁰ Thorek, M.: Large Collection of Foreign Bodies in Stomach. Internat. Clinic., No. III, s. 34.
- 60 Vandivert, A. H., and Mills, H. P.: Jour. A. M. A., vol. lvi, p. 180, January 21, 1911.
- ⁴¹ Winslow, R.: Foreign Bodies in the Stomach. Am. Surg., vol. lxx, pp. 60-64, 3 pl., Philadelphia, 1919.
- ⁶² Wardell, W. H.: Canad. Med. A. Jour., vol. xiv, p. 1105, 1924.
- Eliason, R. L.: A Case Illustrating Foreign Bodies in the Stomach. Jour. A. M. A., vol. lxix, pp. 2106-2107, Chicago, 1917.
- 64 Gussenbauer: Wien. Med. Presse, vol. xxiv, p. 1608, 1883.
- Halsted, W. S.: Johns Hopkins Hospital Reports, p. 1047, 1900.
- ⁶⁸ Warbasse, James P.: Tetany, and foreign bodies in the stomach. Tetany caused by mass in stomach, composed of forty metallic articles, weighing one pound; gastrotomy; cured. Annals of Surgery, Philadelphia, vol. xl, pp. 909-15, 1904.

VON HABERER'S TECHNIC FOR RADICAL STOMACH RESECTION FOLLOWING PREVIOUS GASTROENTEROSTOMY

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The radical resection of the stomach following gastroenterostomy constitutes one of the most difficult operations upon the stomach. During my service as an assistant in the von Haberer clinic, about 20 per cent. of all

stomach cases coming to operation had had a previous stomach operation. In most instances, the previous operation had been a gastroenterostomy. These cases came to the clinic for relief either because of the development of a marginal gastro-jejunal ulcer or because of the failure of a gastric or duodenal ulcer to heal or the patient had developed a recurrent ulcer. In view of Professor von Haberer's vast experience with ulcers and resections, I believe his technic is worthy of description.

The operation of choice in the clinic is a Billroth No. 1 because it is the most physiologic-anatomic operation. By means of the end-to-end anastomosis, the stomach empties directly into the duodenum, whose mucous membrane and alkaline fluids are particularly adapted to handling the acid contents of the stomach. The direction of flow of intestinal contents is maintained and mechanical complications are less to be feared.

Upon opening the abdomen through a median incision, the adhesions between the liver, stomach and duodenum are separated and a careful examination made of both the anterior and posterior walls of the stomach and duodenum. There is no hesitation to open both the gastro-hepatic and gastro-colic omenta. The transverse colon is lifted up and any adhesions freed. The line of anastomosis is closely inspected and palpated. A resection having been decided upon due to the presence of a gastro-jejunal ulcer or ulcers of stomach and duodenum, the adhesions surrounding the gastroenterostomy are separated by blunt and sharp dissection. This is carried

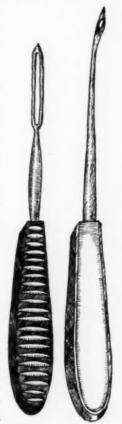


Fig. 1.—Special fine dissecting sound with aneurism needle to match.

out very carefully until a way coursing transversely is made posterior to the loops forming the anastomosis. Great care is necessary to prevent injury of colic vessels. Through this opening posterior to the gastroenterostomy a long sponge forcep is inserted to act as a guide and assist in anatomic orientation. (Fig. 2.)

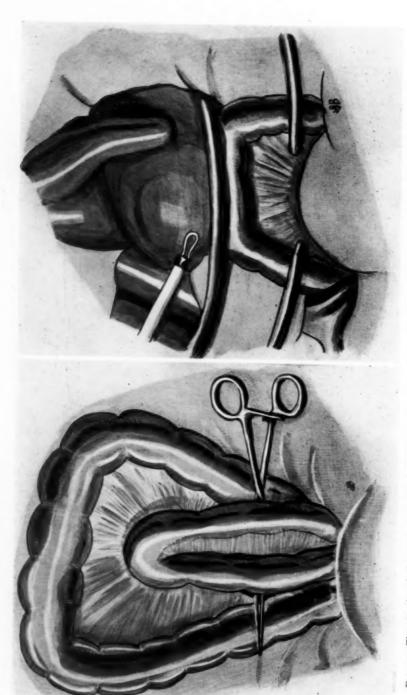


Fig. 2.—The margin of the anastomosis has been freed and a long Fig. 3.—A clamp placed on the line of anastomosis and severance with anstomosis. This helps in anatomic orientation.

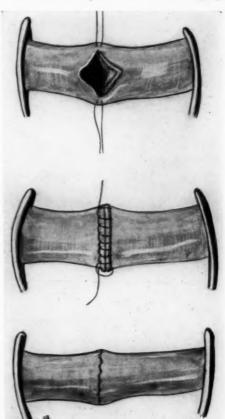
RADICAL STOMACH RESECTION

Through an opening in the gastro-colic omentum and with the transverse colon held slightly elevated, the transverse mesocolon is completely separated from the margin of the anastomosis by working from above and below.

The attention is then turned to the duodenum, which must be made as free as possible. Too much emphasis cannot be placed upon this phase of the operation and success cannot be contemplated unless there is satisfactory mobilization of the duodenum. When the duodenum has not been thoroughly

freed, it is impossible to remove ulcers of the posterior wall. The reported cases of recurrent ulcers after a Billroth No. 1 are in most instances cases where duodenal ulcers have been left on the posterior wall at the time of resection. Professor von Haberer usually starts on the duodenum by ligating the right gastric and right gastro-epiploic arteries. The separation of the duodenum is carried out by the use of the fine instruments shown in Fig. 1. Only small amounts of tissue are taken up at a time and then it is cut between ligatures. The ligations of the gastro-hepatic and gastro-colic omenta are then completed. On the lesser curvature this is carried up to where the left gastric artery enters the stomach wall.

The gastroenterostomy is then taken down. If the line of anastomosis does not show too much infiltration and the jejunum is not too narrow, resection of the jejunum is not required. A curved Doyen clamp is placed upon or just below



F16, 4.—The defect in the jejunum is sutured transversely.

the line of anastomosis. Intestinal clamps are placed upon the jejunum just before and behind the anastomosis to prevent regurgitation of jejunal contents. The anastomosis is severed just below the Doyen clamp by means of an electric cautery. (Fig. 3.) The rent in the jejunum is closed by suturing transversely. (Fig. 4.) Guide sutures are placed at both ends and a continuous locked catgut suture applied to the mucosa. The ends of the suture are left long and replace the former guide sutures. Interrupted linen Lembert sutures are inserted to complete the closure.

If it is apparent that considerable jejunal wall must be removed, a resection of the jejunum is necessary, followed by an end-to-end anastomosis. In

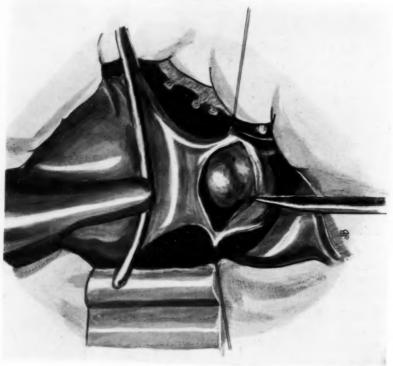


Fig. 6.—The anterior wall has been incised and inspection of the posterior wall shows two ulcers. One ulcer is rather low and with the clamp method probably would not have been seen or removed.

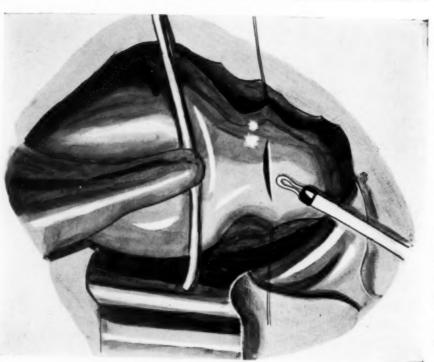
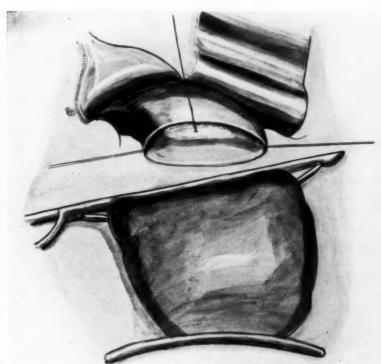
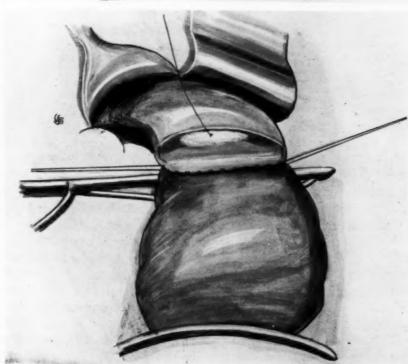


Fig. 5.—The thumb and two fingers grasp the pylorus and guide sutures are placed on the duodenum. The line of incision is below any ulcer that is present.

RADICAL STOMACH RESECTION

Fig. 7.—The posterior wall has been incised and inspection reveals no lower situated ulcers. Small gauze compress in duodenum.





lower Fig. 8,—The stomach is brought over to the duodenum and the interrupted sutures are all inserted before being tied. This allows the stomach to be brought down to the approximate size of the duodenum.

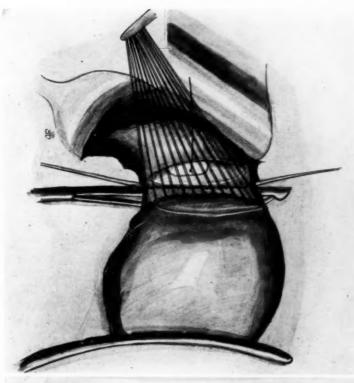
this procedure the mesentery of the jejunum just opposite the anastomosis is ligated. Crushing clamps are placed on the jejunum on either side of the anastomosis. Intestinal clamps are then placed about one inch distant from the crushing clamps. By means of an electric cautery the intestine on either side close to the crushing clamp is severed. An end-to-end anastomosis is made by first inserting a layer of interrupted linen Lembert sutures on the posterior wall and then a continuous locked catgut suture to the mucosa. The anastomosis is completed by the layer of interrupted linen Lembert sutures on the anterior wall. The slit in the mesentery is closed by interrupted catgut sutures.

If necessary and possible, the duodenum is further mobilized by doubly ligating small strands of tissue and cutting. Care must be taken not to injure the posterior wall. At times this cannot be avoided because of bleeding. A Doyen clamp is placed on the pyloric region of the stomach. The pylorus is grasped between the thumb and fingers of the left hand and guide sutures placed on the lower and upper margins of the duodenum at the desired level of severence. (Fig. 5.) Fortunately, in most cases these can be placed below the level of the ulcers. With the pylorus and both guide sutures held at slight tension, the anterior wall of the duodenum is cut through below the ulcers with the electric cautery. Upon opening the anterior wall one is able to make a direct inspection of the mucous membrane of the posterior wall. (Fig. 6.) To prevent regurgitation of duodenal contents, which is always small in amount, a small sponge on a string is inserted into the duodenum. The posterior wall is then cut through below the ulcers and fortunately this can be done in most cases. (Fig. 7.) If no ulcers remain and if the posterior wall has not been too severely traumatized or devoided of peritoneum and not too short, a Billroth No. I is performed.

A clamp is placed on the stomach at the desired level of resection. It has been Professor von Haberer's experience that better results follow fairly wide resections. The clamped stomach is then brought into approximation with the duodenum to determine whether or not the end-to-end anastomosis may be done without too much tension. A linen Lembert suture is then placed through the lower angles of the stomach and duodenum and another through the upper angles of the stomach and duodenum. Midway between these two sutures is inserted another suture through stomach and duodenum. These are not tied but clamped. The two former guide sutures on the duodenum are now removed. Between these three sutures at intervals of about 2 millimetres on the duodenum and naturally at a somewhat greater interval on the stomach are placed linen Lembert sutures. After this row of posterior Lembert sutures has been placed, the two angle sutures and the middle suture are tied. The remaining sutures are then tied and all are cut except a guide suture on either end. (Fig. 8.) The catgut hæmostatic sutures as devised by von Haberer are then inserted in the following manner: The serosa and muscular layers of the stomach are cut through about onehalf inch above the row of interrupted Lembert sutures. A row of inter-

RADICAL STOMACH RESECTION

Fig. 9.—The serosa and muscular layers have been incised and the row of hæmostatic catgut sutures inserted. These sutures also further reduce the size of the end of the stomach.



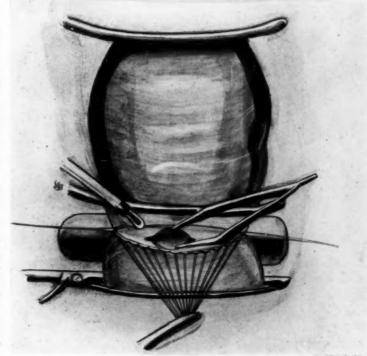


Fig. 10.—The stomach has been thrown to the right and a row of hamostatic catgut sutures inserted on this wall. The sutures have been left long to serve as guide and retraction sutures. The mucosa is being incised with the electric cautery.

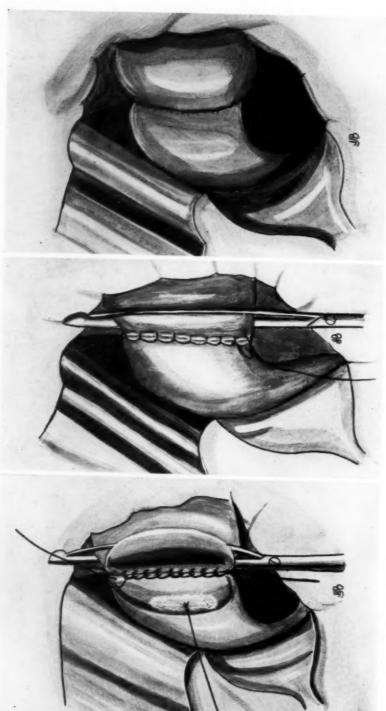


Fig. 11.—The continuous locked catgut suture on the Fig. 12.—The continuous locked catgut suture posterior wall,

Fig. 13.—The anterior row of interrupted linen Lembert sutures. The stomach "mushrooms" down over the duodenum.

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rupted hæmostatic catgut sutures are placed along the lower cut margin. (Fig. 9.) The sutures are not only hæmostatic but also narrow the opening of the stomach. The stump of the stomach is then thrown to the right and after cutting through the serosa and musculature about one inch above the clamp, a similar row of sutures is placed on this surface of the stomach. (Fig. 10.) These two groups of hæmostatic sutures are left long and clamped so that one group is to the right and the other group to the left. They serve as guiding and retracting sutures. A clamp is placed on the stomach just above the row of sutures and the stomach amputated with the electric cautery. (Fig. 10.) The group of sutures to the right are then cut and the mucosa of the posterior wall of the stomach and duodenum sutured. (Fig. 11.) This is a continuous locked catgut suture passing through all layers. Just before turning back on to the anterior wall with the suture, the group of sutures to the left are cut. After continuing the same suture on the anterior wall for a short distance, the small gauze compress in the duodenum is removed and the mucosa suture completed. (Fig. 12.) As on the posterior wall, the interrupted linen Lembert sutures on the anterior wall are first placed at the angles and in the middle. These are immediately tied. The clamp on the stomach is removed and the row of interrupted linen Lembert sutures completed. With this row of sutures, a greater amount of tissue is picked up by the needle on the stomach than on the duodenum. It is remarkable how the stomach "mushrooms" over the duodenum. (Fig. 13.) The lumen usually admits the thumb and two fingers. Professor von Haberer has never seen a stenosis follow his Billroth No. I.

If this modified Billroth No. I cannot be carried out, an anterior Billroth No. II is done with an enteroenterostomy. As compared with the simple posterior Billroth No. II, there is in this procedure less possibility of stasis and back pressure developing in the duodenal stump, and later, should a secondary operation upon the stomach be required, it is much easier to take down an anterior Billroth No. II than a posterior Billroth No. II.

16

LATE RESULTS OF SIMPLE SUTURE IN ACUTE PERFORATION OF DUODENAL ULCER*

BY WILLIAM CRAWFORD WHITE, M.D.

AND

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FROM THE SURGICAL SERVICE OF THE ROOSEVELT HOSPITAL

Acute perforating ulcer of the duodenum was first described by Joseph Penada, of Padua, in 1793. Archibald Malloch²⁸ has given us a most interesting abstract of the case with a reproduction of Penada's plate. Penada described the perforating ulcer as follows:

"Four finger-breadths below the pylorus, i.e., at the commencement of the duodenum, there presented itself to my eye, a very singular oblong hole, resembling an incision made with a knife. (Fig. 1.) It measured eight Parisian lines in length and in breadth about two. The external edge of this cleft, or, one should rather describe it as a peculiar morbid ulcer, was of considerable thickness. To the touch it was sensibly hard and somewhat indurated, and was turned in upon itself in a wart-like fashion, thus indicating that this peculiar local ulceration of the intestine was not of recent origin. The callous lips of this perforation were surrounded by a zone, or rather, a reddened area, which reached out for about an inch around the ulcer, and, shading gradually into a lighter color, extended upwards to the pylorus and for a less considerable distance below the ulceration."

Simple suture was advised by Mikulicz in 1880. Twelve years later the first successful operation for acute perforation was reported by Heussner.²³ In spite of forty years of operative experience since then, opinion as to the operation of choice has not yet been crystallized. Surgeons of skill and experience write very contradictory articles, and these have occasionally appeared in the same number of the same journal. All agree, of course, that the essential factor in mortality is the time allowed to lapse between the time of perforation and the time of operation. The heated differences of opinion bear particularly on the end-results that follow conservative procedures, as compared with those which follow the more radical operations.

Eight years ago, puzzled by the situation, Guthrie²⁰ sent out a questionnaire to many prominent surgeons all over the country, in an effort to find out their opinions in regard to the procedure of choice in the presence of an acute perforation of gastroduodenal ulcer. He received 152 replies to this, and the answers revealed great differences of opinion among the leaders of the profession. This is rather remarkable when one considers the condition as a common abdominal emergency seen not infrequently on all general surgical services.

A review of the literature published since Guthrie's questionnaire reveals,

^{*} Read before the New York Surgical Society, March 11, 1931.

TREATMENT PERFORATED DUODENAL ULCER

instead of a trend toward uniformity of opinion, a still further divergence of the "radical" and "conservative" groups, due to the influence of very radical tendencies in gastric surgery in Europe. This situation furnishes an excuse for the reopening of a much-discussed problem. We have reviewed the literature in the hope of explaining some of the discrepancies in published statistics, and of coming to some conclusion as to the proper interpretation.

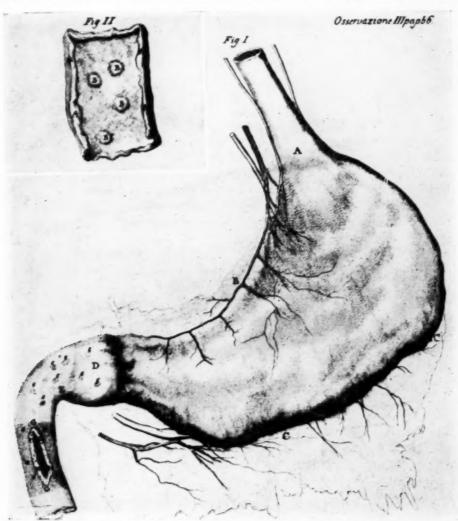


Fig. 1.—Penada's Case of Perforating Ulcer of the Duodenum. (From Malloch: Contributions to Med. and Biolog. Research, dedicated to William Osler, vol. i, p. 137, 1919. Paul B. Hoeber.)

We have also made an effort to follow up some of the old cases at the Roose-velt Hospital. We realize, of course, that the variations in statistics may be attributed partly to the fact that the same type of operation is bound to give different results in the hands of different surgeons. Although we confine ourselves largely to late results, it would seem appropriate to review briefly

some of the more relevant facts concerning the pathology and clinical characteristics of acute perforation of peptic ulcers.

Incidence.—Most of the acute perforations occur in the third, fourth and fifth decades—nearly 40 per cent, in the fourth—although there are recorded instances in which this catastrophe has occurred in very young children and in the later seventies. The outstanding fact of interest in regard to incidence is the extraordinary frequency of the condition in males as compared to females. Less than 3 per cent. of the cases in a very large collected series occurred in females, and it is not uncommon to find a consecutive series of fifty or sixty cases made up, without exception, of males. Although duodenal ulcer of all types is considerably more common in males, the very great difference in the frequency of perforation in the two sexes is difficult to explain, and few have attempted it. Finney16 points to the anatomical fact that the first part of the duodenum is more transverse in females, and, therefore, more constantly bathed in alkaline bile. Others blame "the more active life" of the average man as predisposing to perforation, and also emphasize the excessive use of alcohol as an apparently important factor. One is unable to become enthusiastic over any of these attempts at the explanation of a very puzzling fact.

Symptomatology.—It is doubtful if many acute perforations occur without any previous warning. Undoubtedly, this does happen, but not as frequently as the text-books would lead us to believe. Most of the cases are so acutely ill at the time of admission to the hospital that a past history is not very thoroughly gone into, and then not checked up carefully during convalescence. If this were done, definite antecedent symptoms would be found to have been present in the great majority of cases. However, one of our patients was a well-trained physician, who has assured us that he had never had the slightest subjective evidence of the presence of an ulcer prior to the moment of perforation.

Characteristically, there is a "pre-perforation" exacerbation of a chronic ulcer pain, which represents serosal involvement by the pathological process, which lasts from several hours to two weeks, and is characterized by its constancy and the fact that food and alkalies no longer relieve.

The final inciting cause of the perforation has been a matter to intrigue the interest of many writers. A full stomach and sudden exertion have been considered factors. Meyer³¹ found that nearly four-fifths of a fairly large number of cases occurred more than two hours after a meal, and reports five cases that occurred during sleep. An alcohol spree has also been considered a fairly frequent inciting cause of the actual perforation.

In a series of sixty-two of our cases, only 3 per cent. gave a history of gastro-intestinal bleeding, and in Deaver's series of fifty-five cases, blood was encountered in the stools or vomitus only twice. It is of considerable interest that the ulcers which bleed are not apt to perforate.

We are not concerned primarily with the classical clinical picture that these patients present after perforation, but may we mention in passing that the "shock" so often emphasized in text-books as one of the characteristic features in these cases is not a true surgical shock. "If we use the word to indicate the appearance, and not the state of the patient, then, and only then, can we say that the victim of a perforation suffers from shock." The pulse, temperature and leucocyte count may all be normal in very early cases, but likely to throw one off his guard in this grave emergency. The absence of liver dullness is a late finding that is of no use in early diagnosis. On the contrary, the sub-diaphragmatic gas bubble, seen in vertical X-ray plates, is a great help. Of considerable interest is the supraclavicular pain, usually on the left side, that is said to occur soon after perforation and lasts but a short time (Gibson¹⁸ and Johnston²²). We have been unable to affirm this observation.

In about half of the cases seen in the first twelve hours, vomiting has not occurred at all. In the other half, it has usually occurred only once or twice. In late cases, when the irritating fluid has found its way down the right lumbar gutter into the pelvis, rectal tenderness is usually extreme. This may be of great help in differentiating from other acute *upper* abdominal or thoracic conditions, but it is, of course, a misleading sign when one is attempting to rule out an acute lower abdominal condition. This confusion has led us to make the wrong diagnosis of acute appendicitis a number of times.

Pathology.—We are not concerned with the question of etiological factors in the production of peptic ulcers, nor with "predisposing" factors. Nor are we considering "slow" or "sealed" perforations or "perforating" ulcers, but only the classical free perforations constituting true emergencies. It might be fitting to mention a few points about the pathology of this type before proceeding to the consideration in which we are most interested—that of the late results.

It is not strange that figures as to the relative frequency of perforation on the two sides of the pyloric ring are so variable, as the pyloric vein in these cases is often quite obscured by cedema. In less than 5 per cent. of our own cases did the operator consider the ulcer definitely gastric, rather than duodenal, while some writers (Farr, 15 Dunbar 11) consider that "most" of them are really gastric. The important fact in the matter is that the vast majority of the ulcers that perforate are so situated anatomically that a simple infolding operation may produce an occluded pylorus. Just how likely this is to occur will be discussed in full when we consider the arguments in favor of the routine performance of gastroenterostomy in these cases.

Two questions of considerable interest pathologically are these: (1) What is the usual length of time that an ulcer has existed prior to perforation? (2) Given a patient with a duodenal ulcer, what is the percentage chance of an acute perforation? To question No. I there can be no definite answer. There is great difference of opinion on the second problem, and most of the opinion is based on inadequate data. Frequently, one finds in the literature a dogmatic statement that "most of these perforations are of chronic ulcers"—while it is easy to find equally dogmatic statements insisting

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that they are acute. It is our opinion that the perforation practically always takes place on the site of a chronic ulcer, as may be demonstrated from the examination of such ulcers as we have excised. The best opinion in regard to the second question seems to be that the usual patient with duodenal ulcer is taking only an extremely small chance of perforation—not enough to add weight to the surgical side of the controversy as to the treatment of chronic duodenal ulcer.

Nearly all of the perforations, as we have seen, have occurred in the first four centimetres of the anterior wall of the duodenum and have presented a cleanly punched-out perforation usually three to six millimetres in diameter, and surrounded by a considerable zone of induration. The posterior ulcers, of course, adhere to the pancreas or some other neighboring surface before the stage of perforation is reached, and therefore do not give rise to the calamity of acute free perforation. It has been suggested by many observers that perforated and non-perforating ulcers differ pathologically. Along with many other pathologists and surgeons, we do not agree with this attitude but consider that perforation is merely one of the several possible serious complications of any typical peptic ulcer. However, as we have already mentioned, ulcers that bleed seem very unlikely to perforate.

Just why many of these ulcers should heal permanently after simple closure is somewhat puzzling. Apparently, the centre of the ulcer is extruded and the surrounding protective zone of induration heals promptly and permanently. Evidence of the complete disappearance of the ulcer is plentiful. There are many reports in the literature of subsequent abdominal operations or autopsies at intervals varying from three months to eighteen years after a simple closure of a perforated ulcer, at which time not the slightest trace of the original pathology remained (Lecene, ²⁵ Basset, ³ Brenner, ⁴ Pannett ⁴⁰).

Our excuse for discussing the pathological traits of these perforated ulcers in a paper that purports to deal entirely with late surgical results lies in the apparent plausibility of the opinion that perforating and non-perforating ulcers present different surgical problems, not only immediately, but remotely as well. The good results that followed cautery-puncture and closure of many chronic duodenal ulcers intimate that we might consider acute spontaneous perforation as a part of the "cautery-puncture" operation, made-to-order, as it were, and apt to be followed by good and permanent healing if promptly closed.

Choice of Operation.—There are so many factors to be considered here that it seems wise to list some of the *pros* and *cons* of each of the advocated procedures, and then to draw what conclusions seem justified by the actual facts that are available.

Simple Suture.—A great many leading surgeons are confident that simple closure of the perforation is the procedure of choice. The suture material, and the exact method of using it may differ slightly, but the principles to which these surgeons point are as follows: They claim:

- (1) That it is the quickest, simplest procedure to meet a grave emergency, and that it adequately meets the emergency.
 - (2) That the operative mortality is lower than with any other procedure.
 - (3) That the post-operative course is smoother.
- (4) That the ulcer, in the majority of cases, heals promptly and remains healed.
- (5) That subsequent pyloric obstruction is infrequent, and that when it does occur there is an ideal indication for a secondary gastroenterostomy under the most favorable conditions. "Two safe operations are always better, and to be desired, than one too dangerous."²⁰
- (6) That primary gastroenterostomy subjects the patient unnecessarily to the dangers of gastrojejunal ulcer.
- (7) That gastroenterostomy does not prevent the occasional occurrence of hæmorrhage, reperforation, formation of new ulcers, or the recrudescence of old ones.
- (8) That a careful evaluation of late results justified the above impressions.

Closure Plus Immediate Gastroenterostomy.—There are many advocates of this procedure, most of whom agree that it should be done only in comparatively early cases. They make the following claims:

- (1) That gastroenterostomy does not increase the mortality in early cases, adding only fifteen minutes to the procedure.
- (2) That the danger of "spreading infection" by the additional procedure has been overemphasized, the exudate in these cases being almost uniformly sterile for many hours after perforation.
- (3) That the immediate post-operative course is much smoother, with fluids given orally much sooner than is possible after simple closure.
- (4) That the perforation cannot, in many cases, be safely and adequately closed without producing a pyloric obstruction of considerable degree.
- (5) That the ulcer heals rapidly in the presence of a gastroenterostomy, and that complications such as reperforation are less apt to occur.
 - (6) That the late results are better than with simple suture.
- (7) That a large percentage of simple suture cases require subsequent gastroenterostomy.

Pyloroplasty.—Such operative procedure as the Heineke-Mikulicz, Finney, Horsley, and other modifications of pyloroplasty have at times been quite popular in treating acute gastroduodenal perforations. The advocates of such procedures point out:

- (1) That peptic ulcers which present acute perforations are usually situated just where such an operation can be easily done—on the anterior surface near the pylorus.
- (2) That the operation takes only a few minutes longer than simple closure, and does not involve any soiling of the lesser peritoneal cavity, as in gastroenterostomy.
 - (3) That the operation serves the purpose of avoiding the possibility of

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future pyloric stenosis, without subjecting the patient to the "unphysiologic" procedure of gastroenterostomy.

(4) That the lesion is directly attacked, and actually removed.

Resection.—Advocates of immediate partial gastrectomy in these cases vary in the operation they refer to—the extreme being a sort of miniature Billroth No. I operation on one hand, and a sub-total gastrectomy on the other.

They would limit the operations to those cases that reach the surgeon soon after perforation, but claim that in these cases the rather formidable procedure does not add greatly to the operative mortality. They emphasize that patients who have the more usual and conservative procedure done at the time of acute perforation require subsequent surgery in a rather high percentage of cases. They believe that the doing of a really curative operation, rather than a palliative one, should be the aim of the surgeons even in the presence of an acute perforation.

Discussion.—Although the same facts are interpreted differently by the different groups, a review of all available facts bearing on this problem would seem so important as to quite overshadow the theoretical discussion of pros and cons that we have listed. It has been our endeavor to find out what actually happens to this group of patients.

Gibson, 18 who has long been interested in this subject, did simple closure in seventy-five of seventy-six cases of acute perforation. Half of the cases remained symptom-free, and in only eight cases did the symptoms indicate the necessity of a secondary operation. Farr¹⁵ found that nineteen of twenty-one cases remained well after simple closure, and ten of Brenner's twelve cases remained symptom-free.⁵ Two-thirds of Pool's⁴³ series of simple closure cases were cured by the procedure. Urrutia⁵⁵ recently reported a series of fifty-two private patients in whom he had done simple closure of acute duodenal perforations. Of this series 63.6 per cent. remained well. Southam⁴⁹ reports that pyloric obstruction eventually necessitated a gastroenterostomy in only four of thirty-seven cases of simple closure. These rather optimistic reports are bolstered considerably by the opinion of such men as Finney,16 Pannett,40 and Engelsing,12 who have considered the results of simple closure very satisfactory. Johnston²² collected results in 568 cases of simple closure, and 25 per cent. of these had persisting symptoms. Only 11 per cent. required gastroenterostomy later. Williams and Walsh⁵⁶ report that of fifty-eight cases with simple suture, subsequent gastrojejunostomy was necessary in only ten. Lewisohn²⁷ classes thirteen of thirty-three cases followed as "failures" from conservative surgical treatment. Mills³² thought the late results of simple closure very discouraging.

On the basis of available statistics it seems fair to estimate that of every 100 cases that leave the hospital following simple closure of an acute perforation, approximately sixty to sixty-five will remain free of gastric symptoms, approximately ten to fifteen will require further surgery, and of the remaining twenty-five those who are reasonably careful about diet and general activity will get along satisfactorily.

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In addition to clinical evidence, we have pathological evidence to support the statement that many ulcers heal (and remain healed) after simple closure. There are numerous cases to be found in the literature in which subsequent exploration for some other reason revealed that there was no trace of the old ulcer (Lecene, ²⁵ Basset, ³ Brenner, ⁴ Pannett ⁴⁰). An experimental support for the view that pyloric obstruction does not occur as commonly as would be supposed, in cases where a perforation near the pylorus has been infolded, is to be found in the work of Stewart and Barber ⁵¹ on dogs. They performed cautery puncture of the duodenum near the pylorus in a series of dogs, and infolded the opening so completely that the lumen was practically occluded. X-ray and clinical studies subsequently seemed to show that it is very difficult to produce pyloric obstruction in this way. The same conclusion was reached by Eliot, ¹⁴ who found that the constriction produced by excision of two-thirds of the circumference of the duodenum and closure (in cats) ironed out very promptly.

Our own experience with the late results of simple suture has been so encouraging that we continue to do this operation as the routine procedure in these cases. We have been able to follow nineteen of these cases for a prolonged period—all of them having been carefully checked up at frequent intervals. Thirteen of these have remained well, the average interval since operation being more than five years. One of these cases is uncomfortable after heavy indulgence in alcohol. The others are all symptom-free, some of them in spite of careless diet.

Of the remaining six cases, one developed pyloric stenosis two months following operation and required gastroenterostomy. A second case was well for eight years, then began to vomit and lose weight and had a partial gastrectomy while in Germany. The third case, who has been followed for seven years, is comfortable only when very careful about his diet. The others have been troubled by very characteristic ulcer symptoms. One of these latter three suffered a recurrence of symptoms five and one-half years after operation, during which interval the patient had been entirely well. We interpreted this as likely due to a new ulcer.

In a larger series of sixty-two cases, reported by the junior author at a staff conference several years ago, it was found that approximately 10 per cent. of the cases treated by simple suture required a subsequent gastroenterostomy.

The advisability of instituting drainage in these cases finds also a surprising difference of opinion. Gibson¹⁸ found that cultures from the exudate were almost invariably sterile up until eighteen hours after perforation, and many observers have pointed out the widespread nature of the soiling, and the futility of attempting to drain all the affected area. The question of drainage in late cases finds all in agreement that it should be done, the only disagreement being as to the details. Nearly all the answers to Guthrie's²⁰ questionnaire agreed on drainage—many of them advising a suprapubic drain as well as subhepatic drainage. Deaver¹⁰ and Brown⁶ urge the use of a

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suprapubic drain as very important. Finney¹⁶ advises against the use of any drainage except in late cases, and urges multiple thorough drainage if any. We have for some years past been closing the early cases without drainage, and have been very much pleased with the results, most of the wounds healing per primam.

Nearly thirty years ago, Movnihan35 suggested that it might be wise to add some procedure, after closure of an acute perforated ulcer near the pylorus, which would guard against the future development of a pyloric stenosis. Since that time it has become more and more common for the surgeon to perform a pyloroplasty with excision of the ulcer or an immediate gastroenterostomy after closing the perforation. In many hospitals, the latter is done as a routine in early cases. The advocates claim that the mortality is actually decreased by the additional procedure. They suggest that a pyloric perforation is often inadequately closed because of fear of producing an obstruction by too much infolding, and that if the perforation is thoroughly closed and infolded an ideal condition for the function of a gastroenterostomy is produced. There appeared in the literature so many statements to the effect that gastroenterostomy lowers the mortality that Cope,8 a careful student of acute abdominal surgery, wrote a letter of protest to the British Medical Journal to give it as his opinion that this was due to the selection of cases. He referred to an analysis of five years at St. Mary's Hospital, London, during which time the mortality of simple suture cases was higher than in those cases in which closure of the perforation was combined with gastroenterostomy, pointing out, however, that the time elapsing between perforation and operation in the former group was thirteen hours, and in the second group only five and one-half hours. The same observation applies to Dineen's44 cases. Ten cases were subjected to immediate gastroenterostomy, with no deaths, but the average time elapsing before operation was only three and three-quarter hours.

"The statistics of the advocates of suture and gastrojejunostomy are not comparable with those of surgeons who advise suture only. Gastrojejunostomy is generally carried out in selected cases only, and the statistics of a series of such cases are, therefore, not comparable with a series when suture only has been done, for the latter would include the bad as well as the good operative risks. In many of our cases where, from the general condition of the patient, and the duration and the size of the perforation, the case would have been judged fit to withstand a gastrojejunostomy, we have been impressed with the critical condition they have passed through before convalescence has been established. We cannot but feel that in many of these cases, the additional strain of a gastrojejunostomy would have turned the scales against them."

Mortality Record of the Roosevelt Hospital

Time after operation	Number	Deaths	Per Cent.
Under 10 hours	50	3	6
10 to 20 hours	13	6	46
20 to 30 hours	10	4	40
Over 30 hours	6	4	66
	-	_	
	79	17	21.5

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Of these cases, fifty-three were simple suture with ten deaths, an operative mortality of 19 per cent. Twenty-six cases had simple suture plus immediate posterior gastroenterostomy with seven deaths, an operative mortality of 27 per cent.

In spite of these latter figures, we feel that gastroenterostomy at an early stage has not, on the average, such a high mortality as we report here. Three of our gastroenterostomies were performed in cases that had been perforated over twenty hours and these three patients died. We consider that in these cases poor surgical judgment was used regardless of a conservative or a radical attitude.

Seven cases, not included in the above table, were subjected to an immediate excision of the ulcer with pyloroplasty. These were all early cases except one which was performed late, and this patient was the only one to die.

An important consideration is that of the late results in those cases subjected to immediate gastroenterostomy, as compared to those treated by simple suture. Only six of our cases were followed over three years and of these four were considered cured. Johnston²² collected a series of 157 cases. Only 12 per cent. of these had "severe" persisting symptoms, and the percentage that required operation for gastrojejunal ulcer was 1.3. Mills³² had ten out of fourteen perfectly well.

Cutler and Newton⁹ decided, after a careful review of the literature and of their own cases, that it is wiser to do a gastroenterostomy as a routine in these cases. However, Metzger³⁰ concluded, after a review of late results in nearly a hundred cases, that the late results are about the same whether gastroenterostomy is added to the closure of the perforation or not.

Lewisohn,27 who strongly advocated routine gastroenterostomy a few years ago, has more recently stated that he considers the likelihood of gastrojejunal ulcer so great that gastroenterostomy should not be done. In early cases he advises partial gastrectomy. McCreery²⁹ urges picking the operation to fit the case, and feels that the best late results will follow simple suture in the majority of cases, although certain cases, where the perforation is large and the induration is extensive, definitely call for immediate gastroenterostomy. Unlike most European surgeons, Urrutia⁵⁵ is very strong for conservatism in these cases. He concludes that "gastroenterostomy does not modify the post-operative course—is an insufficient treatment of the ulcer itself, and may have unfortunate late results." His opinion becomes more valuable when one realizes that he has had a most extensive experience with gastric surgery, having performed more than 500 partial gastrectomies for chronic gastric and duodenal ulcers. In spite of his dexterity and radical tendencies in "elective" gastric surgery, he does not feel that any procedure should be added to a simple closure in the presence of acute perforation,

Noehren³⁸ quotes McKnight, of the Mayo Clinic, that in seven of twenty re-explored cases, the ulcer had persisted or new ones formed in spite of the presence of a gastroenterostomy. Radoïevitch⁴⁵ collected more than 10,000

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cases of gastroenterostomy for a duodenal ulcer, and eighty-one of these cases perforated in spite of the presence of a gastroenterostomy. There are frequent reports of reperforation, formation of new ulcers, etc., after gastroenterostomy in these cases, that give one the definite impression that there should be a trend away from gastroenterostomy except in cases of established pyloric obstruction of considerable degree.

Pyloroplasty, of the Heineke-Mikulicz or Horsley type, has been discussed earlier in this paper as an ideal theoretical solution to the problem of acute perforated peptic ulcers. Unfortunately, the late results do not justify this hope. Erdmann,57 who did many of these operations for chronic ulcer some years ago, has come to the conclusion that the late results of this operation are poor. Hinton²¹ reported the Horsley type of pyloroplasty in twelve cases with one death (the time after perforation varying from four to fifteen hours). Nine of the survivors were traced (sixteen months to four years) and only two of these were entirely well after three years, seven results being unsatisfactory. One of Lecene's25 cases was well for six years after excision of the ulcer and pyloroplasty, and then had severe recurrence of pain. Williams and Walsh⁵⁶ report five of six pyloroplasty operations that had good end-results. Rowlands and Turner46 report twelve with only seven good results. Our own experience with the follow-up results in six cases has not been encouraging. X-ray studies seemed to indicate that too-rapid emptying was responsible, in part at least, for the persisting symptoms.

In his monograph on gastroduodenal ulceration, Pannett⁴⁰ refers to the advocacy among European surgeons of the performance of partial gastrectomy in the presence of an acute perforation as "an amazing development." Even more amazing, in view of the fact that subtotal gastrectomy in elective operations carries even in the most expert hands a mortality of 10 per cent. to 16 per cent., are the low mortality figures given for this method of treating acute perforations.

The theoretical considerations have already been discussed. As to the actual results of gastric resection, these are not available, for the mounting list of reports in the literature nearly all represent recent cases. The radical tendency in European gastric surgery may be illustrated by the experience of one of our own cases. He had been entirely symptom-free for more than two years following the simple closure of a perforated duodenal ulcer. While on a recent visit to his home in Graz, he was advised to have a subtotal gastrectomy done at once, on the grounds that anyone who had ever had a duodenal ulcer would never be permanently well until subjected to partial gastrectomy.

ODELBERG³⁰ reports twenty partial gastrectomies in acute perforations with only one death. All were early cases. The results of the cases traced more than a year were good. He believes in spite of the pain and the "emergency" atmosphere, most of these early cases are in better condition to stand a major gastric operation than the average patient who has had a prolonged siege of dieting on account of chronic ulcer. The only death in his series occurred in a case in which cultures from the peritoneal exudate showed a hæmolytic streptococcus.

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Krainik⁵⁰⁸ adds his testimony on the side of resection, and refers to the first successful resection done in France in the treatment of acute perforation, in 1919. Kunz⁵¹⁴ reports seven resections without a death, two of the cases having been perforated more than fifteen hours. Radoïevitch⁵¹⁵ gives a mortality figure for resections done within the first twelve hours after perforation as 11.29 per cent. One of Lewisohn's⁵¹⁷ partial gastrectomies (out of four done for persisting symptoms after simple closure) developed a marginal ulcer. One of Lecene's⁵¹⁵ cases met the same fate, and recent reports of Balfour²¹ at the Mayo Clinic warn us that the incidence of gastrojejunal ulcer following partial gastrectomy is a very definite one.

CONCLUSIONS

1. In the vast majority of cases of acute perforated gastroduodenal ulcer, including those that reach the surgeon soon after perforation, simple closure of the perforation, with or without drainage, is the treatment of choice. In early cases the abdomen should be closed without drainage.

2. Patients so treated who survive the immediate hazard have approximately a 60 per cent. to 65 per cent. chance of remaining permanently quite free of gastric complaints, and a 10 per cent. to 15 per cent. chance of requiring a gastroenterostomy at a later date on account of pyloric obstruction.

3. The remaining 25 per cent. will be comfortable if careful as to diet and general daily routine. Some of them might best be subjected to partial gastrectomy as a curative measure.

4. Secondary gastroenterostomy, done on account of subsequent pyloric obstruction after simple closure, should produce uniformly good results. Primary gastroenterostomy does not produce uniformly good results and should not be done as a routine.

5. A few cases of acute perforated gastroduodenal ulcer are seen in which adequate closure cannot be effected without producing a pyloric occlusion, on account of a large perforation, unusually extensive surrounding induration, or both. These cases may justifiably be subjected to immediate gastroenterostomy. Careful closure with use of omental reinforcement will reduce the cases in which this is necessary to a minimum.

6. The late results of the Horsley type pyloroplasty in these cases are only fair.

7. Partial gastrectomy in the presence of an acute perforation is not justified.

BIBLIOGRAPHY

- Ball, W. G.: Perforated Gastric and Duodenal Ulcers. St. Bartholomew's Hosp. Reports, vol. lix, pp. 49-95, 1926.
- ² Balfour, D. C.: Surgical Clinics of North America, p. 1420, December, 1927.
- ⁸ Basset, A.: Les Résultats éloignés dans les ulcères perforés de l'estomac et du duodenum. Bull. and Mém. Soc. Nat. de Chir., vol. 1, pp. 224-232, 1924.
- ⁴ Brenner, E. C.: Perforated Ulcers of the Duodenum: a Report of Fifteen Consecutive Cases. Surg., Gynec., and Obst., vol. xxxiv, p. 370, 1922.
- ⁶ Brenner, E. C.: Perforated Ulcers of the Duodenum: a Study of Twenty-seven Cases. Annals of Surgery, vol. lxxxvi, pp. 393-400, 1927.
- ⁶ Brown, K. P.: The Late Results in a Series of Forty-nine Cases of Perforated Gastric and Duodenal Ulcers. Edinburgh M. J., n.s. xxxii, pp. 207-213, 1925.

WHITE AND PATTERSON

- ⁷ Brutt, H.: Das perforierte Magen—und Duodenalgeschwur. Ergebn. d. Chir. u. Orthop., vol. xvi, pp. 516-576, 1923.
- ⁸ Cope, Zachary: British Medical Journal, p. 139, January, 1925.
- ⁹ Cutler, E. C., and Newton, F. C.: Perforated Ulcer of the Stomach and Duodenum, Boston M. and S. J., vol. clxxxviii, p. 789, 1923.
- ²⁰ Deaver, J. B., and Pfeiffer, D. B.: Gastroenterostomy in Acute Perforating Ulcer of the Stomach and Duodenum. Annals of Surgery, vol. lxxiii, pp. 73, 441, 1921.
- ¹¹ Dunbar, J.: Acute Perforating Peptic Ulcer: an Analysis of 287 Cases. Glasgow M. J., vol. cvi, pp. 109–113, 1926.
- ¹² Engelsing, H.: Zur Frage der Behandlung der akuten Magen und Duodenal perforation. Deutsche Ztschr. f. Chir., vol. clxxxvi, pp. 25-40, 1924.
- Evans, A. J.: Operative Treatment of Acute Perforated Ulcer of the Stomach and Duodenum: Observations on Sixty-seven Cases. British M. J., vol. i, p. 184, 1926.
- ¹⁴ Eliot, E., Corscaden, J. A., and Jameson, J. W.: Clinical Features and Treatment of Acute Perforating Gastric and Duodenal Ulcer. Annals of Surgery, vol. lv, p. 546, 1912.
- ¹⁵ Farr, C. E.: Perforating Gastric and Duodenal Ulcer. Annals of Surgery, vol. 1xxii, pp. 591-594, 1920.
- Finney, J. M. T.: The Surgery of Gastric and Duodenal Ulcer. Amer. J. Surg., n.s. i, pp. 323-343, 1926.
- ¹⁷ Gay, Pierre: A propos du traitement des ulcères perforés gastro-duodénaux . . . et en particulier les résultats obtenus par la suture simple. Paris Thèse, No. 532.
- ¹⁸ Gibson, C. L.: Acute Perforations of Stomach and Duodenum. Boston M. and S. J., vol. clxxxix, pp. 425-433, 1923.
- ¹⁹ Gibson, C. L.: Acute Perforations of Stomach and Duodenum. Amer. J. M. Sc., vol. clxv, pp. 809-816, 1923.
- ²⁰ Guthrie, D.: Should Gastroenterostomy Be Performed in the Presence of Ruptured Duodenal Ulcer? N. Y. State J. Med., vol. xxiii, p. 66, 1923.
- ²¹ Hinton, J. W.: Surg., Gynec., and Obst., vol. xlvii, p. 407, 1928.
- ²³ Johnston, L. B.: Acute Perforation of Gastric and Duodenal Ulcers. Internat. Clinics, 36 s, vol. ii, pp. 145-156, 1926.
- ²⁵ Krainik, R.: Contribution à l'étude du traitement de l'ulcère perforé gastrique et duodénal. Paris, 1922.
- ²⁴ Kunz, H.: Ueber das perforierte Magen-duodenalgeschwür und das perforierte ulcus pepticum jejuni. Arch. f. klin. Chir., vol. cxl, pp. 419-426, 1926.
- Eccene: Résultats éloignés du traitement des ulcères gastro-duodénaux perforés. Bull. et Mém. Soc. Nat. de Chir., vol. 1, p. 275, 1924.
- ²⁶ Lewisohn, R.: Late Results in Perforated Gastroduodenal Ulcers. Annals of Surgery, vol. lxxxvii, pp. 855-860, 1928.
- ²⁷ Lewisohn, R.: Persistence of Pyloric and Duodenal Ulcers Following Simple Suture of an Acute Perfor ation. Annals of Surgery, vol. lxxii, pp. 595-590, 1920.
- Malloch, Archibald: An Early Record of Perforating Duodenal Ulcer. Contributions to Medical and Biological Research. Dedicated to Sir William Osler. Vol. i, pp. 137–145, 1919. Paul B. Hoeber.
- McCreery, J. A.: Acute Perforated Ulcer of the Stomach and Duodenum. Annals of Surgery, vol. clxxix, pp. 91-99, 1924.
- ³⁰ Metzger, A.: Contribution à l'étude des ulcères gastro-duodénaux perforés en péritoine libre, envisagés à deux points de vue. Paris Thèse, No. 346, 1925.
- ^{at} Meyer, K. A., and Brams, W. A.: Acute Perforation of Gastric and Duodenal Ulcer: a Study of Sixty-two Consecutive Cases. Amer. J. Med. Sc., vol. clxxi, pp. 510–514, 1926.
- ²² Mills, G. P.: The Treatment of Perforated Gastric and Duodenal Ulcers; with a Report on Forty Consecutive Cases. Brit. M. J., vol. i, p. 12, 1925.
- ³⁸ Moynihan, Lord: Addresses on Surgical Subjects. W. B. Saunders, 1928.

TREATMENT PERFORATED DUODENAL ULCER

- ^{ns} Moynihan, Lord: Perforation of Gastric and Duodenal Ulcer. Practitioner, vol. exx, pp. 137-174, 1928.
- 35 Moynihan, Lord: Lancet, vol. ii, p. 1662, 1901.
- Muhsam, R., and Unger, E.: Ueber das perforierte Magen-und Duodenalgeschwur. Arch. F. Verdauungskr., vol. xxxvii, pp. 140-156, 1926.
- ⁸⁷ Naumann, H.: Kritische Bemerkungen ueber den heutigen Stand der Therapie des frei perforierten Magen-Zwölffingerdarmgeschwürs. Arch. f. klink. Chir., vol. cxxxix, pp. 434-470, 1926.
- ⁵⁶ Noehren, A. H.: Acute Perforation of Gastric and Duodenal Ulcer. Amer. J. Surg., vol. xxxviii, pp. 59-64, 1924.
- Odelberg, A.: Resection of the Stomach in Perforating Ulcer. Acta Chir. Scandin., vol. lxii, pp. 159-166, 1927.
- ⁴⁰ Pannett, C. A: The Surgery of Gastroduodenal Ulceration. London, Oxford Univ. Press, 1926.
- ⁴ Pauchet, V.: Perforations aiguës de l'estomac et du duodénum. Paris Chir., vol. xv, pp. 441-446, 1923.
- ⁴² Perrin: Résultats éloignés de la suture des perforations des ulcères gastro-duodénaux (avec ou sans gastro-entérostomie). Lyon Chir., vol. xxi, pp. 499-502, 1924.
- ⁴³ Poll, E. H., and Dineen, P. A.: Late Results of Gastroenterostomy for Gastric and Duodenal Ulcers, Including Perforated Ulcers. Annals of Surgery, vol. lxxvi, pp. 457-466, 1922.
- "Dineen, P. A.: Acute Perforated Ulcers of the Stomach and Duodenum. Annals of Surgery, vol. xc, p. 1027, 1929.
- ¹⁵ Radoïevitch, S.: Du meilleur traitement chirugical des ulcères gastro-duodénaux en péritoine libre. Rev. de Chir., vol. Ixiii, pp. 161–212, 1925.
- 46 Rowlands and Turner: Operations of Surgery, Seventh Ed., vol. ii, p. 117, London.
- ⁴⁷ Schwarz, E.: Ueber die operative Behandlung des perforierten Magen-Duodenal-Geschwürs. Deutsche Ztschr. f. Chir., vol. cxcii, 239-265, 1925.
- ⁴⁵ Smith, F. H.: Diagnosis and Treatment of Perforated Duodenal Ulcer, Founded on Forty-one Cases. Brit. M. J., vol. ii, pp. 1068–1070, 1921.
- ⁴⁹ Southam, A. H.: The Treatment of Perforated Gastric and Duodenal Ulcer. Brit. M. J., vol. i, p. 556, 1922.
- Eo Stenbuck, J. B.: Causes of Death Following Operations for Perforated Gastric and Duodenal Ulcer. Annals of Surgery, vol. lxxxv, p. 713, 1927.
- ¹⁴ Stewart, G. D., and Barber, W. H.: Acute Perforating Ulcer of the Stomach or Duodenum. Annals of Surgery, vol. 1xxv, pp. 349-355, 1922.
- 52 Troutt, M.: Surg., Gynec., and Obst., vol. xlvii, p. 6, 1928.
- ES Turner, G. G.: The Perforation of Gastric and Duodenal Ulcer and After. Lancet, vol. i, pp. 183-185, 1925.
- ⁶⁴ Urrutia, L.: Sur le traitement opératif des ulcères gastro-duodénaux perforés. Paris Chir., vol. xv, pp. 243-247, 1923.
- ⁵⁶ Urrutia, L.: Late Results in Perforated Gastroduodenal Ulcers. Annals of Surgery, vol. xl, p. 73, 1929.
- 66 Williams, H., and Walsh, C. H.: Lancet, vol. i, p. 9, January 4, 1930.
- ⁶⁷ Erdmann, J. F.: Personal Communication.

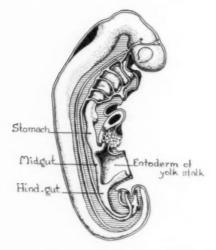
PERFORATED PEPTIC ULCER OF MECKEL'S DIVERTICULUM

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MECKEL's diverticulum is a congenital abnormality having definite surgical significance. It is subject to a variety of pathologic conditions. Its presence is but rarely suspected pre-operatively yet it may present a clinical picture of grave importance and one demanding immediate attention.

Johann Meckel (1781-1833) discovered this diverticulum and described its anatomic origin. Its presence is due to the persistence of a portion of the



4.2 human embryo (His. Prentiss) x25

Yolk stalk
Allantoic stalk
Cloaca

5 mm. human embryo (His). x 25

F16. 1.—Section of human embryo at two weeks. Note the large opening of the yolk stalk and its close relationship to the stomach and pancreas.

F1G. 2.—Section of human embryo at four weeks. The yolk stalk has become much smaller.

vitelline duct or yolk stalk. In the human embryo the yolk sac is but a rudiment of the important organ found in some lower animals. At the end of the second week of fœtal life this yolk sac is connected with the primitive gut by a wide opening, the yolk stalk (Fig. 1). During the third and fourth weeks this yolk stalk becomes much constricted (Fig. 2) and by the sixth week of fœtal life there remains only a small patulous cord extending from the gut to the umbilicus (Fig. 3). Normally, this yolk stalk undergoes complete regression. Its failure to degenerate might result in a variety of conditions. By remaining patulous throughout there would be a fecal fistula discharging at the umbilicus. A complete obliteration of its lumen might result in a fibrous cord extending from the gut to the navel, a potential source of intestinal obstruction. The duct might remain patent only at the periphery and give rise to an umbilical fistula. Obliteration of the duct

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at both ends might result in a cystic tumor or enterocele, which could be situated anywhere along the fibrous cord from the ileum to the umbilicus, or the enterocele may be contained within the mesentery or within the walls of the ileum. The persistence of the iliac end of this structure resulting in a pouch connecting with the lumen of the gut is what is commonly spoken of as Meckel's diverticulum.

The incidence of Meckel's diverticulum is low. Operating-room statistics

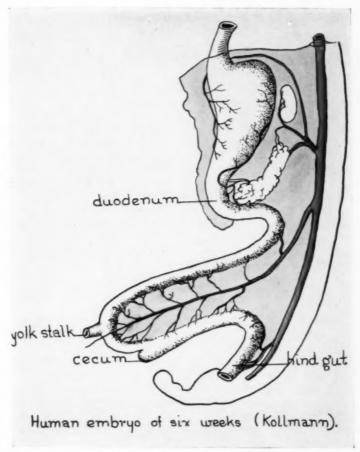


Fig. 3.—Gastro-intestinal tract of human embryo at six weeks. The yolk stalk has become constricted to a patulous cord.

are not accurate, as a rule, because few surgeons make a routine search for its presence. At the Johns Hopkins Hospital, 15 cases were found out of 2,600 necropsies, or 5 per cent., while Mitchell, of Chicago, out of 1,635 autopsies reported 39 cases, or 2.25 per cent. It occurs twice as often in males as in females.

Meckel's diverticulum is usually located along the terminal three feet of the ileum. It may vary in size from a small out-pouching to a large viscus filling one-half the abdominal cavity, as in the case recently reported by Yates.

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It might result in intestinal obstruction by wrapping itself around and constricting the gut or by becoming invaginated into the lumen of the ileum producing an intussusception. Being a rudimentary blind pouch it is subject to the same disease processes which have brought fame and dishonor to the vermiform appendix—inflammation, gangrene and perforation. Foreign bodies might become lodged within its lumen and perforate its walls, as reported by Berry. Meckel's diverticula, not infrequently, are lined totally or in part by gastric mucosa, thus making them the seat of peptic-ulcer formation with subsequent hæmorrhage and perforation. Following is the report of such a case:

L. G., a well-developed boy of eighteen years, was pursuing his usual occupation on the farm when he became suddenly blind, fainted and shortly afterward passed a large amount of dark blood from his bowels (estimated at about a quart). His past history was negative. At the age of nine years he had an attack of abdominal pain which was diagnosed by a physician as appendicitis and treated expectantly. He had been well otherwise except that for six months preceding his present illness he had had an occasional substernal gnawing sensation which was unrelated to the time of eating but was relieved by taking soda. The day following the onset of his trouble he became nauseated and vomited twice. There was no blood in the vomitus. That day he also passed a small amount of very dark blood from his bowels. He was placed under the care of Dr. C. H. Rand, with whom I saw him that night. In the poorly lighted farm house the boy appeared extremely ill. He responded to questions very poorly or not at all. The skin was cool, the temperature subnormal, the pulse rate 130 and of poor volume. Physical examination was otherwise negative except for slight tenderness along the right side of the abdomen. A positive diagnosis was not reached but it was felt that he probably had a bleeding duodenal ulcer. He was removed to the hospital in order that he might be given a transfusion and other supportive treatment and his condition studied more thoroughly. When he arrived two hours later he seemed to be much improved. The skin was warm and his temperature normal. The pulse rate was 100 and of fair volume. He was comfortable and abdominal palpation revealed very little tenderness along the right side. Still adhering to the original provisional diagnosis he was given a hypodermic of morphine, an ice cap was placed upon the abdomen and he was allowed to go through the night. The following morning his temperature had risen to 99°, the pulse rate was 106, there was definite tenderness and rigidity in the right lower quadrant, the tenderness extending somewhat across the lower abdomen. Rectal examination disclosed marked tenderness throughout his pelvis. Urinalysis was negative. Examination of his blood showed 80 per cent. hæmoglobin, 4,375,000 red cells, 18,000 leucocytes and 72 per cent, polymorphonuclears. It was then felt that he had appendicitis, the appendix having ruptured since his admission to the hospital.

A right rectus incision was made. On opening the peritoneum there was seen a small amount of old blood. The appendix was found to be situated retrocecally. It was not acutely inflamed and was not the cause of the patient's symptoms. It was removed and the stump inverted. The terminal ileum was then explored. A Meckel's diverticulum was found about 45 cubic millimeters from the ileocecal junction. The diverticulum was about 5 cubic millimeters in length and 3 cubic millimeters in diameter at its base, tapering to a tip. There was definite evidence of acute and chronic inflammation about the diverticulum. Several of the adjacent coils of the ileum were bound into the inflammatory process by a plastic exudate and the diverticulum was densely adherent to one side of the ileum. The base of the diverticulum was opposite the mesenteric attachment of the ileum. (Fig. 4.) On separating these structures a perforated ulcer about ½ cubic millimeter in diameter and having a punched-out appearance was found just

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above the base of the diverticulum. The remainder of the diverticulum was not involved by the diseased process. (Fig. 5.) The diverticulum was resected, using a rubber-covered clamp on the ileum and the opening in the ileum was closed transversely, using two rows of continuous chromic gut suture and reinforcing with a Lembert suture. This left a satisfactory lumen in the ileum. No further exploring was done. Two Penrose cigarette drains were placed in the pelvis and the incision closed.

Convalescence was uneventful. The boy left the hospital on the fourteenth day after the operation. When seen again two months later he appeared in excellent health.

Pathologic examination by Dr. J. B. Bullitt.—The serosal coat is very thick; it is not covered by endothelium but by a sort of hyaline material; apparently this has been adherent to some neighboring viscus. The muscle coat is well developed in part of the specimen, very thin in part of it. The mucosa is not like the typically normal mucosa of either stomach or gut. A small portion has villi and the straight tubules like the gut; most of the epithelium in this is of the goblet-cell variety. The greater part of the mucosa has crypts like the stomach, and rather tortuous glands. The epithelium in the crypts is entirely goblet cells, while that in the tubules is almost a continuous layer of oxyntic cells. The perforation is just at the junction of the two types of mucosa. There is very little inflammatory exudate about the ulcer. There is moderate leucocytic infiltration (chiefly lymphoid) throughout serosal coal. (Fig. 6.)

Etiology.—In a careful study of thirty specimens of Meckel's diverticulum Schaetz found that about 50 per cent. contained abnormal elements. The mucosa may resemble that of the jejunum or duodenum. Pancreatic tissue was found in 10 per cent. He places the incidence of gastric mucosa in Meckel's diverticulum at 16 per cent. He proposes the theory of embryonal transplantation as an explanation for these findings. Certainly in early feetal life the yolk stalk, the anlage of Meckel's diverticulum, is in close proximity to the pancreas and fore-gut. (Fig. 3.) A Meckel's diverticulum may be lined entirely with gastric mucosa, as in the cases reported by Magevand and Hübschmann or the gastric mucosa may be scattered in islands throughout the diverticulum. According to Cuibal a Meckel's diverticulum may act as a miniature stomach and the acid secretions corrode the junction of the gastric and intestinal mucosa, thus producing an ulcer by the same processes as a peptic ulcer of the jejunum. Meulengracht is of the same opinion and believes there is no doubt that the ulcer is a true peptic ulcer, being produced by the stagnation of gastric juice at the floor of the diverticulum. Aschner and Karelitz have recently made an exhaustive review of the literature on this subject and have collected thirty-three cases of proven and probable peptic ulcer of Meckel's diverticulum. To this number can be added one case reported by Etchegorry, two by Feure, Patel and Lepart and the author's case, in all of which gastric mucosa was demonstrated at the site of the ulcer. This brings the total of reported cases to thirty-seven. The age of the patients ranged from five months to forty-five years, 70 per cent. occurring in those of fifty years and under. Eighty-six per cent. of the cases were males. Etchegorry states that simple ulcers of Meckel's diverticulum have been overlooked until recent years.

Symptoms.—In only one of these cases is there a record of a correct preoperative diagnosis having been made. This is the case reported by A. S.

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Fig. 5.—Diverticulum separated from ileum "Punched-out" ulcer near base of diverticulum.



Fig. 4.—Appearance of Meckel's diverticulum at operation. Plastic exudate has been thrown out about the diverticulum and adjacent coils of ileum.

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Jackson in which the diagnosis was first made by the family physician, Dr. E. A. Ketterer, of Montfort, Wis. Appendicitis was the diagnosis most often made, this being followed in frequency by peritonitis of unknown origin, intussusception, duodenal ulcer, intestinal polyp and tuberculosis. Intestinal hæmorrhage was the most frequent complication, this having occurred in 30 cases or 81 per cent. The bleeding is often profuse and may be the first indication of any trouble, or the patient may pass a number of small bloody stools over a period of several weeks. The blood may be bright red or it may have undergone partial decomposition within the intestine and appear as a black tarry stool, as though the hæmorrhage had taken place high up in the gastro-intestinal tract. There is usually some associated abdominal

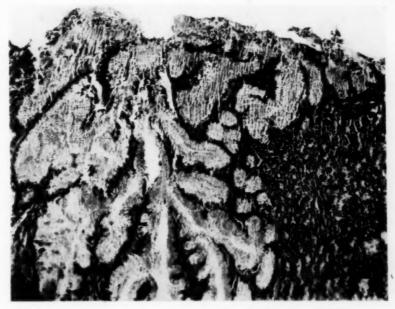


Fig. 6.—Photomicrograph of section near ulcer. Epithelium resembling that of gastric mucosa.

discomfort although it does not always have the typical text-book location in the right lower quadrant and radiate to the umbilicus. Magevand's case had severe abdominal pains for a period of three years. These pains occurred before meals and were relieved by food. The author's case complained of a substernal gnawing sensation that was relieved by soda. Next to hæmorrhage the most frequent complication in peptic ulcers of Meckel's diverticulum is perforation. This occurred in twenty-one cases, or 56 per cent. Not one of these cases was correctly diagnosed. The contents of the lower portion of the ileum being more septic than that of the stomach and duodenum, it is natural to suppose that a perforation in this location would be more dangerous. Eight of these cases died following operation or the condition was discovered at autopsy, giving a mortality in the perforated group of 38 per cent.

Treatment.—Any Meckel's diverticulum possesses numerous possibilities for serious trouble. It would seem that all cases of severe or repeated intestinal hæmorrhage in which other causative lesions have been excluded should be explored for the possible presence of a peptic ulcer in a Meckel's diverticulum. The frequency of perforation and the high mortality following this complication argue against undue procrastination. Resection of the diverticulum is the procedure of choice. This may have to be extended to remove a portion of the ileum, if the ulcer is situated at the base of the diverticulum, or if the ileum itself is involved by the ulcerating process.

BIBLIOGRAPHY

- ¹ Abt, I. A., and Strauss, A. A.: Meckel's Diverticulum as a Cause of Intestinal Hæmorrhage. J. A. M. A., vol. lxxxvii, pp. 991–995, September 26, 1926.
- ^a Aschner, P. W., and Karelitz, S.: Peptic Ulcer of Meckel's Diverticulum. Annals of Surgery, vol. xci, pp. 573-582, April, 1930.
- ⁸ Berry, J. A.: Perforation of a Meckel's Diverticulum. British Journal of Surgery, vol. xv, p. 331, October, 1927.
- Cuibal, L.: Peptic Ulcer of Meckel's Diverticulum Causing Profuse Intestinal Hæmorrhages; Operation; Cure. Bullet. Mem. Soc. Nat. de chir., Paris, vol. 1, p. 349, 1924.
- ⁵ Etchegorry, J.: Perforation of Simple Ulcer of Meckel's Diverticulum; Operation; Recovery. Anales de le Focultad de Medicina, vol. xi, pp. 621–628, 1926.
- 6 Hallopeau and Humbert: Generalized Peritonitis Due to Perforation of an Ulcer of Meckel's Diverticulum, Presenting the Aspect and Characters of a Simple, Socalled Peptic Ulcer. Med. Inf., vol. xxx, p. 229, Paris, 1924.
- 7 Hübschmann: Late Perforation of a Meckel's Diverticulum After Trauma. Muenchener Midizinische Wochenschrift, vol. ix, pp. 2051–2053, September 16, 1913.
- * Jackson, A. S.: Ulcer of Meckel's Diverticulum as a Cause of Intestinal Hæmorrhage. Annals of Surgery, vol. lxxxv, pp. 252-256, February, 1927.
- ⁹ Fevre, M., Patel, M., and Lepart.: Ulceres Perfores du Diverticule de Meckel, Bull. et Mem. Soc. Nat. de chir., vol. lvi, pp. 756-767, 1930.
- et Mem. Soc. Nat. de chir., vol. lvi, pp. 750-767, 1930.

 ¹⁰ Kleinschmidt, Karl: Peptic Ulcer of the Meckel Diverticulum, Bruns Beitrage Zur Klinischen Chirur., vol. cxxxviii, pp. 715-720, 1927.
- ¹¹ Peterman, M. G., and Seeger, S. J.: Meckel's Diverticulum with Hæmorrhage, Transactions of the Association of Resident and Ex-resident Physicians of the Mayo Clinic, vol. ix, pp. 154–157, 1928.
- Mayo, W. J., and Johnson, A. C.: Meckel's Diverticulum. Surgical Clinics of North America, pp. 1127–1130, October, 1926.
- ¹³ Magevand, C., and Durant, R.: Peptic Ulcer of Meckel's Diverticulum. Rev. de chir., vol. xi, p. 536, Paris, 1922.
- Meulengracht, E.: On a Meckel's Diverticulum Which Was Partly Lined with Gastric Mucous Membrane and Which Was the Seat of a Peptic Ulcer. Virchow's Archiv., vol. ccxxy, pp. 125-128, 1918.
- 15 Schaetz, G.: Quoted by Aschner.
- ¹⁶ Stulz, E., and Woringer, P.: Peptic Ulcer of Meckel's Diverticulum. Annals of Surgery, vol. Ixxxiii, No. 4, pp. 470-478, 1926.
- ¹⁷ Wolfson, W. L., and Kaufman, B.: Acute Inflammation of Meckel's Diverticulum. Annals of Surgery, vol. 1xxxiv, No. 4, pp. 535-540, April, 1929.
- Yates, H. B.: A Remarkable Meckel's Diverticulum. The British Journal of Surgery, vol. xvii, pp. 456-462, January, 1930.

THE SIMILARITY IN EFFECT OF EXPERIMENTAL HIGH INTESTINAL OBSTRUCTION AND HIGH COMPLETE INTESTINAL FISTULA*

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FROM THE UNIVERSITY OF ROCHESTER SCHOOL OF MEDICINE

For several years now, a debate has been going on between investigators engaged in the study of intestinal obstruction. It is generally conceded that there is a wide difference between strangulation and simple obstruction. In strangulation every one agrees that toxemia is a prominent feature, but in simple obstruction there has been a difference of opinion. One group believes that toxemia plays an important rôle here also; but the other workers hold that the loss by vomiting of fluids, salts, and the products of normal secretions from the upper intestinal canal is responsible for the train of symptoms produced. Several ingenious methods of experiment have been designed to preserve these materials in obstructed animals. The resulting long survival and good condition of these obstructed animals lend weight to the argument against the toxemia theory.

It seemed to us that a crucial test of the nontoxic hypothesis could be offered by comparing the picture resulting from the loss of the upper intestinal secretions through vomiting in obstructions at a given point with that associated with an equally effective loss of these secretions through complete intestinal fistula at the same level. If there is no toxic absorption from the obstructed loops involved, but simply an emptying of their secretions, there should be no difference in the effect produced, whether the emptying be through the stomach or through a fistula. Accordingly, such comparisons were made in the following experiments.

Twenty-four albino rats were completely obstructed below the level of the biliary and pancreatic ducts by tying a tape across the bowel. This point measured one and one-half inches (3.8 centimetres) from the pylorus. The average time of survival was found to be thirty hours, with extremes of twenty hours and sixty hours.

Twenty-four albino rats had the intestine divided at the same level. The lower end in each instance was tied off with a tape, but the upper end was sutured with a wide open mouth to the cutaneous surface, establishing a complete fistula. The average time of survival was found to be thirty-three hours, with extremes of twenty hours and sixty hours. The results of these survival experiments are shown in Chart 1. Ether anæsthesia was used throughout. There was no evidence of peritonitis in either series.

Six dogs were obstructed at a point ten inches (25.4 centimetres) below

^{*} Presented before the University of Rochester Medical Society, February 16, 1931.

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the duodeno-jejunal ligament by dividing the bowel at this point and turning in the ends under purse-string sutures. The average time of survival was

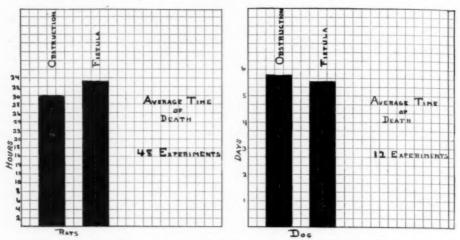


Chart 1.—The average time of death in twenty-four rats with obstruction is thirty hours. The average time of death in fistula at the same place is thirty-three hours.

Chart 2.—In dogs the animals with fistula died sooner than those with obstruction. A reversal of the picture in rats.

five and four-fifths days. Six dogs had complete fistulas established at the same level, the upper end of the divided bowel in each instance being sutured

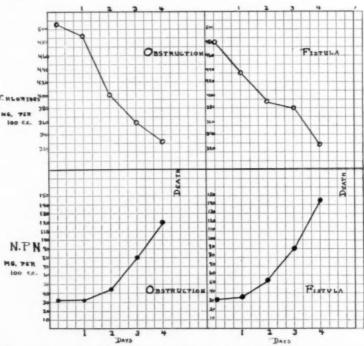


Chart 3.—An example of the blood-chemistry changes in dogs with obstruction and fistula at the same level. The similarity in the picture is very striking.

with wide-open mouth to the cutaneous surface. The average time of survival of these animals was five and one-half days. (Chart 2.) Ether

RESULTS OF INTESTINAL OBSTRUCTION AND OF FISTULA

anæsthesia was used throughout. There was no evidence of peritonitis in either series.

Comparisons between the essential blood-chemical changes were made in the dogs with complete fistulas and those with obstruction. The nonprotein nitrogen and the sodium-chloride readings were practically identical in the two series. Individual charts of two animals bringing out this similarity are shown in Chart 3.

The table of the chemical changes in the blood of the dogs with complete jejunal fistulas is here appended. (Table I.) The tables of the chemical changes in the blood of the dogs with complete jejunal obstructions at the same level were previously published.¹ The nonprotein nitrogen of the whole blood was determined by the method of Folin and Wu, and the chlorides by a modification of the method of Van Slyke.

Table I

Complete Fistula Ten Inches Below Duodeno-jejunal Ligament

Blood Amount per 100 cubic centimetres

	Total		
	Day After	Nonprotein	Chlorides
Dog No.	Operation	Nitrogen, Mg.	Mg.
30-214	0	39.8	462.2
	I	64.2	310.1
	2	152.8	257.4
	3	De	ead
30-194	0	32.0	379-7
	I	35-4	438.8
	2	54.2	392.0
	3	89.6	380.3
	4	146.4	327.6
	5	De	ead
30-161	0	33-4	456.3
	1	39.2	409.5
	2	41.8	339.3
	3	49.0	339.0
	4	71.6	280.8
	5	not rec	corded
	6	128.0	234.0
	7	D	ead
30-197	0	38.2	456.3
	I	36.2	409.5
	2	45.2	386.1
	3	120.0	327.6
	4	D	ead
30-215	0	37.0	514.8
	1	46.2	444.8
	2	53.6	427.1
	3	60.0	468.0
	4	86.0	384.0
	5	170.0	375.0
	6	300.0	350.0
	7	De	ead

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30-168	0	39.8	464.9
	I	39.0	432.9
	2	40.0	386.1
	3	42.8	409.0
	4	46.6	374-4
	5	58.0	352.0
	6	80.0	385.0
	7	177.0	335.0
	8	Dea	ad

Discussion.—A consideration of the results obtained in these experiments makes it seem likely that the clinical picture produced is indistinguishable in complete simple high obstruction and complete fistula at the same level. If there is a toxemia present, which may still be argued on account of the rise in nonprotein nitrogen, it must equally be present in the two conditions. This makes it obvious that the obstruction per se is not the essential factor in producing the picture. The syndrome, both clinical and chemical, is associated with loss of the contents of the involved intestinal tract and not with their retention. Clairmont and Ranzi,² Braun and Borrutau,³ and Enderlen and Hotz,⁴ have all shown that absorption comes practically to a standstill in the loops of obstructed intestine. Consequently, in obstruction the contents of the distended loops of bowel and stomach may contain the essential material, but it cannot be used.

Much information is already available as to the importance of different constituents of the secretion of the upper gastro-intestinal tract. The investigations of Haden and Orr,⁵ Gamble, et al.⁶ and others have clearly established the importance of the sodium-chloride content. The total loss of gastric juice has been shown by Dragstedt and Ellis⁷ to produce a similar condition with marked alkalosis. Elman⁸ has deviated the pancreatic secretion so that it was no longer available with striking changes indicating the necessity for this material.

Werelius⁹ contends that the danger stage in obstructions is reached when the bile is no longer secreted; and Brockman¹⁰ has supplied bile per rectum successfully in the treatment of intestinal obstruction.

Experiments have been done by White and Fender,¹¹ Pearse ¹² and others wherein the whole content of the obstructed segment of the gastro-intestinal tract which is normally lost in vomiting or stagnation in the loops is resupplied so that it is made available for the animals. Under such conditions, all evidence of toxæmia disappears, and the general health remains normal for a considerable time. After long periods, a metabolic change of some kind occurs so that there is a loss of weight, loss of appetite and weakness noted.

Changes in the blood chemistry in duodenal fistuals were previously recorded by Walters, Kilgore and Bollmann.¹³ Their work showed chemical changes in the blood which were strikingly similar to those noted in high obstructions. These investigators attached importance to the distension of the duodenum found in animals having a high intestinal fistula. In our

experiments a slight gaseous distension of the duodenum was observed at post-mortem examination, but no evidence of retention was found. This was considered a post-mortem change. The importance of preserving the contents of the obstructed loops has already been noted by Haden and Orr, who claimed a more rapid death after enterostomy in their obstructed animals. It seems probable that there is still a limited field for the use of enterostomy in releasing the tension on the intestine, thus preventing strangulation. Excessive distension is more dangerous than is generally recognized, even though it has received considerable attention in experimental work (Van Zwalenburg, Brooks, et al., Gatch, Trusler and Ayers, Morton, etc.). The duodenal tube for drainage is often successful for the same reason. A much safer method for a bad obstruction would seem to be an internal anastomosis between the obstructed and collapsed loops of bowel, diverting the contents around the obstruction and preserving the essential constituents. The writers have used this method successfully in a number of cases.

The correlation between simple obstruction and complete fistula at the same level is so remarkable that it is strange that there has been so little comment on the similarity. From evidence such as we have presented, it is necessary to assume that either there is a toxæmia present in both, or that there is no toxæmia in either. The search for the toxin has been conducted for so long, with such conflicting results, and with so little evidence that it exists anywhere in the body except in the loop contents, the preservation of which has proved to be so valuable, that it would seem more satisfactory to take the second alternative.

CONCLUSIONS

- (1) Simple high intestinal obstruction and complete fistula at the same level are essentially similar in clinical picture, blood-chemical changes, and life expectancy.
 - (2) Toxamia must be present in both or not present in either.
- (3) In any case, obstruction $per\ se$ is not the important factor, for the picture is produced from loss of the contents of the obstructed bowel and not from their retention.
- (4) The obstructed loop contents have been in the past the only available source for the supposed toxin. No one has demonstrated such a toxin in the blood or other tissues of the obstructed animals.
- (5) It seems more reasonable to conclude, then, that there is no toxæmia in either simple high obstruction or complete fistula.
- (6) The syndrome is due to a deficiency of essential secretions lost from the upper gastro-intestinal tract.
- (7) This may come about by loss through vomiting, or by deficiency in absorption from the disturbed segment.
- (8) Practically every case of intestinal obstruction should be regarded as a potential strangulation.

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- (9) Enterostomy, if too complete, may have a very deleterious effect on intestinal obstruction.
- (10) Measures to preserve the loop contents, such as internal anastomosis, would seem to be more valuable.

BIBLIOGRAPHY

- Morton, J. J., and Stabins, S. J.: Relation of Bacillus Welchii Antitoxin to the Toxæmia of Intestinal Obstruction. Arch. Surg., vol. xvii, p. 860, 1928.
- Clairmont, P., and Ranzi, E.: Zur Frage der Autointoxication bei Ileus. Arch. f. Klin. Chir. vol. lxxiii, p. 696, 1904.
- ³ Braun, W., and Borrutau, H.: Experimental-Kritische Untersuchungen uber den Ileustod. Deutsche Ztschr. f. Chir., vol. xcvi, p. 544, 1908.
- ⁶ Enderlen and Hotz: Ueber die Resorption bei Ileus and Peritonitis. Mitt. a. d. Grenzgeb. der Med. u. Chir., vol. xxiii, p. 755, 1911.
- ⁵ Haden, R. L., and Orr, T. G.: The Effect of Sodium Chloride on the Chemical Changes in the Blood of the Dog After Pyloric and Intestinal Obstruction. J. Exper. Med., vol. xxxviii, p. 55, 1923.
- ⁶ Gamble, J. D., and McIver, M. A.: Body Fluid Changes Due to Upper Intestinal Obstruction. J.A.M.A., vol. xci, p. 1589, 1928.
- ⁷ Dragstedt, L. R., and Ellis, J. C.: The Fatal Effect of the Total Loss of Gastric Juice. Am. J. Physiol., vol. xciii, p. 407, 1930.
- *Elman, R., and Hartmann, A. F.: The Cause of Death Following Rapidly the Total Loss of Pancreatic Juice. Arch. Surg., vol. xx, p. 333, 1930.
- Werelius, A.: Is Death in High Intestinal Obstruction Due to Liver Insufficiency? J.A.M.A., vol. lxxix, p. 535, 1922.
- ¹⁰ Brockman, R. S.: Toxæmia of Acute Intestinal Obstruction. Lancet, vol. ii, p. 317, 1927.
- White, J. C., and Fender, F. A.: The Cause of Death in Uncomplicated High Intestinal Obstruction. Arch. Surg., vol. xx, p. 897, 1930.
- Pearse, H. E., Jr.: Is Toxæmia the Cause of Death in Uncomplicated Intestinal Obstruction? Annals of Surgery, vol. xciii, p. 915, 1931.
- ¹⁸ Walters, W., Kilgore, A. M., and Bollmann, J. L.: Changes in the Blood Resulting from Duodenal Fistula. J.A.M.A., vol. lxxxvi, p. 186, 1926.
- ¹⁴ Haden, R. L., and Orr, T. G.: Effect of Jejunostomy on Experimental Obstruction of the Jejunum of the Dog. J. Exper. Med., vol. xliii, p. 483, 1926.
- ¹⁵ Van Zwalenburg, C.: Strangulation Resulting from Distention of Hollow Viscera, Annals of Surgery, vol. xlvi, p. 780, 1907.
- ¹⁶ Brooks, B., Schumacher, H. W., and Wattenberg, J. E.: Intestinal Obstruction: An Experimental Study. Annals of Surgery, vol. lxvii, p. 210, 1918.
- ¹⁷ Gatch, W. D., Trusler, H. M., and Ayres, K. D.: Causes of Death in Acute Obstruction: Clinical Application and General Principles of Treatment. Surg., Gynec., and Obst., vol. xlvi, p. 332, 1928.
- ¹⁸ Morton, J. J.: The Differences Between High and Low Intestinal Obstruction in the Dog. An Anatomic and Physiologic Explanation. Arch. Surg., vol. xviii, p. 1119, 1929.

DEEP LIPOMAS OF THE HAND

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Deep lipomas about the hand and foot are very seldom identified before operation. Out of thirty-four such cases in the literature only one is stated to have been recognized as such before incision. It is somewhat surprising at first to discover this when subcutaneous lipomas have outstanding characteristics which place them among the most easily recognized of lesions. The difficulty becomes more comprehensible when other factors are considered. First among these is the extreme rarity of the condition. Secondly, lipomas in a tense and inelastic envelope such as a heavy aponeurosis give a somewhat different sensation to the palpating finger than they do under the yielding skin. Last, there is a marked similarity of superficial physical characteristics between a deep lipoma and the very much more common compound ganglion.

Anatomically, the deep lipomas can be divided into groups according to their relationship to the tendon sheaths. The term endovaginal was applied to the largest group by Rohmer¹ to indicate those whose origin and expansion occur within a tendon sheath. This group was still further divided into "lipoma simplex symmetricum" and "lipoma arborescens." The terms are self-explanatory. Billroth was the first to report the occurrence of lipoma arborescens in a tendon sheath. A second group of these tumors may be called epivaginal or subaponeurotic. The first term seems more descriptive since in the cases described as also in my case the growth was attached quite definitely to the tendon sheath rather than to the other subaponeurotic tissues. Both endovaginal and epivaginal lipomas have been described in the wrist, in the palm of the hand, and in the fingers. Endovaginal lipomas have also been described in the dorsum of the wrist and hand, and in the foot.

In the diagnosis of deep tumors of the hand and foot we are limited to consideration of those of mesothelial origin excepting, of course, the traumatic epithelial cysts which occur when epidermis is carried as a free graft into the subcutaneous tissue of the hand. The latter will not be confused with lipomata because of the history of injury and the relatively spherical outline of the cyst. Of the mesothelial group, the only neoplasms possessing physical characteristics likely to be confused with lipomata are the cavernous hemangioma and the cavernous lymphangioma. The hemangioma may be told by its color and its change in size under pressure over the tumor and under obstruction of the venous return in the arm. I have been able to find no record of a deep cavernous lymphangioma occurring in these locations. Such a lesion would be difficult to differentiate if it occurred.

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Fig. 1.



Fig. 2.



Fig. 3.

Figs. 1 and 2.—Epicaginal lipoma beginning at base of 4th fleror tendon sheath.

Fig. 3.—Extension distally along lumbrical tendons.

A more common error lies in confusion of a lipoma with a hygroma of a tendon sheath—most commonly with tuberculous tenosynovitis. The similarity of the two lesions has been so great (especially in the type of tuberculous synovitis that is accompanied by extensive rice body formation) that it has been suggested that lipoma arborescens is a peculiar reaction to tuberculous infection rather than a neoplasm. There is no histologic nor bacteriologic support for this hypothesis; but it is noteworthy that two of the eighteen cases collected by Strauss' were later shown to have tuberculous lesions elsewhere in the body, and Klotz's case had a history of old pulmonary tuberculosis.

Filho states he was able to make a correct pre-operative diagnosis, because he had just read Renfrew White's10 report, and had the possibility of lipoma in mind. In most cases, familiarity with the differing characteristics of tuberculous synovitis and deep lipoma will make the differentiation possible. Crepitation may occur in other lesion and was present in four out of twenty-one cases of arborescent lipoma, with ten authors not recording its presence or absence. Fluctuation, or a strong suggestion of fluctuation may be present in both and was recorded in nine out of twenty-one cases of tendon-sheath lipoma, with six not reporting its presence or absence.6 The distension will follow the outline of a tendon sheath in both lesions; but the swelling of a lipoma may stop abruptly in the middle of the course of a sheath and the lipoma be so attached that pressure will not squirt it from end to end of the sheath as will occur with rice bodies and a tuberculous exudate. complete transfer of the contents of a distended sheath from above to below an annular ligament is possible in a compound ganglion, but not with an attached arborescent lipoma. In many of the cases, the duration of the swelling has been longer than is usual in a tuberculous synovitis without sinus formation-twenty years in one instance. Aspiration has been done many times, but has proved of little aid in diagnosis. In cases of lipoma its only evidence has been negative, and as such has not been of material assistance.

Gosselin quoted in makes the interesting suggestion that lipomata when chilled become of a stiffer consistency; so that application of an ice-bag is a method of differentiating fatty from other types of tumors. Unfortunately, I did not use this in the present case, but I have since employed it in a case of lipoma simplex symmetricum of the extensor tendons of both wrists. In the latter instance, chilling the hands definitely stiffened the consistency of the tendon-sheath enlargements. Treatment by excision, including the attached portions of the tendon sheaths, has yielded excellent results. In some cases this has involved a very extensive and careful dissection to spare tendons and nerves surrounded by or traversing the growth. In the case of Martin and Grenier and in this case incomplete previous removal was followed by a recurrence. Such an accident is somewhat likely when we consider the marked arborescence of some of these tumors.

There have been twenty-one endovaginal lipomas of the hand and foot reported (Strauss, White, 10 Filho6). There have been eleven cases of such

tumors in fingers. Some of the latter were endovaginal and others epivaginal (Martin and Grenier, Muller). There have been three epivaginal lipomas of the mid-palmar space, quite similar to this one in their tendency to follow lumbrical tendons, and also to bulge through the thin areas of the deep palmar fascia.^{7,8,9}

Case Report.—The patient was a healthy man of thirty-four. He was married and had two children. Since childhood there had been a swelling present on the palm of the right hand. At first he believes it was rather small, but it grew slowly and steadily. When he was eight an attempt was made to remove the swelling and an incision was made. However, the mass soon reappeared and has slowly grown to its present size. It has never been painful, and has never interfered with the strength of the fingers or hand. It has, by the mere mechanical presence of its bulk, interfered with the use of his hand as a small cushion would. Occasionally he noted a sensation of tingling and numbness in the ring finger, but he could not localize the sensation within the finger.

Extending from the middle of the palm almost to the distal interphalangeal joint of the fourth finger was a soft, fluctuant swelling. This was roughly the size of a flattened lemon. The dorsum of the fourth finger from the base to the centre of the middle phalanx was broadened and distended to about twice normal width. There was no crepitation, and the sense to the palpating finger was similar to that made by fluid confined in small loculi. Motions of the flexor tendons did not appreciably affect the mass. The skin was nowhere attached to the growth, and finger motions were not impeded except that the size of the palmar mass prevented full closure of the fist. Sensation was normal.

Under ethylene anæsthesia a Martin bandage was applied to the forearm. A longitudinal incision was made over the palmar aspect of the fourth finger from the distal interphalangeal joint to mid-palm. A branching, lobulated lipoma was found beneath the deep palmar fascia. The growth was spread out so that it filled the space between the flexor tendons of the third and fifth fingers. It bulged into the interdigital fossa at either side of the ring finger. It was attached to the flexor tendon sheath of the fourth finger at its palmar terminus. The attachment at this point was very marked, and the neoplasm seemed to project a short distance into the tendon sheath (or originate within the sheath and project outwards). The anterior aspect of the fourth tendon sheath was removed with the tumor. The mass followed the lumbrical tendons dorsally as they ran toward the dorsum of the ring finger, and thus made the swelling on the back of the finger. The digital vessels and nerve on the radial side of the fourth finger tendons passed through the growth and were divided. The vessels were ligated and the nerve ends approximated with two fine sutures. The digital vessels and nerve to the ulnar side of the fourth finger tendons were freed from the mass without injury. Elsewhere the growth shelled out quite easily, although the skin of the dorsum of the finger was completely undermined by the removal of the tumor and it seemed doubtful whether it would be left with an adequate blood supply. The skin and palmar fascia were closed together with interrupted sutures.

Microscopic sections of the neoplasm showed it to be made of adult fat cells with rather more fibrous and vascular tissue than is seen in most subcutaneous lipomas.

BIBLIOGRAPHY

- ¹ Gross, Rohmer, Vautrin and Andre: (quoted by Filho). Nouveaux Elements de Pathologie Chirurgical, vol. i, p. 392, 1900.
- ² Muller, P.: Palmar Lipoma of Finger. Bull. et Men. Soc. de Chir. de Paris, vol. xx, pp. 390-392, May 4, 1928.
- ⁸ Martin, A., and Grenier, J.: A Case of Lipoma of a Finger. Paris Medical, vol. xii, pp. 303-304, April 8, 1922.

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- Strauss, A.: Lipoma of the Tendon Sheaths. Surg. Gynec. and Obstet., vol. xxxv, pp. 161-171, March 18, 1922.
- 5 Klotz, T. Schefellar: Lipoma of Palm. Nederl. Tijdschr. v. Geneesk., vol. ii, p. 4537, September 15, 1928.
- Filho, Brandão: Lipoma Arborescens of Tendon Sheaths. Chirurg. degli Organo di Movimento, vol. ix, pp. 235-244, March, 1925.
- ⁷ Auvray: Lipoma of Palm of Hand. Bull. et Mem. Soc. Nat. de Chirurg., vol. lii, pp. 47-48, 1926.
- Mouchet, A.: Bull. et Mem. Soc. Nat. de Chir., vol. lii, p. 48, 1926.
- Desbonnets: Journ. des Science Med. de Lille, vol. xliv, p. 281, October 31, 1926.
- White, Renfrew: Arborescent Lipoma of Tendon Sheaths. Surg. Gynec. and Obstet., vol. xxxviii, p. 489, 1925.
- ¹¹ Lehman, E.: Deep Lipomas. Journal of Am. Med. Assn., vol. lxxxiv, pp. 1033-1034, April 4, 1925.
- 12 Schulte, F.: Deep Lipoma of Forearm. Med. Klin. vol. xxiii, p. 679, May 6, 1927.

FOOT INFECTIONS OF PERIDIGITAL ORIGIN

ROUTES OF SPREAD AND METHODS OF TREATMENT BY MANUEL GRODINSKY, M.D.

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For the most part, foot infections have been considered more or less en masse with little effort to determine accurately their anatomic location and the common routes of spread. As a result, therapy has likewise been inaccurate and unsatisfactory. In two previous studies (Grodinsky, 1929, 1930), an attempt was made to determine the anatomic background of fascialspace and tendon-sheath infections in the foot by methods similar to those used by Kanavel (1921) in his studies of hand infections. As in the hand, there is in the foot still a third type of infection which may exist alone or may be combined with either fascial-space or tendon-sheath infections. This is the type, usually due to the streptococcus, which spreads by lymphatic vessels producing lymphangiitis, lymphadenitis and, in unfavorable cases, septicæmia and even death. This kind of infection is most serious as regards immediate mortality, but, once the crisis is passed, is least apt to cause permanent disability. On the other hand, fascial-space and, more particularly, tendon-sheath infections, though much less serious to life, are much more liable to leave permanent loss of function.

The purpose of this paper is to describe a series of infections, all originating in the same part of the foot, the region of the toes; to trace the routes of spread; and to discuss the type and results of the therapy applied. This group of cases represents a clinical entity in which there is a common starting place and, in general, common routes of spread. They differ chiefly in the degree to which they have progressed before resolution begins or death occurs.

Etiology.—In most of the cases, the infection started in wounds or abrasions between the toes, on the plantar surface of the toes, or on the ball of the foot adjacent to them. Glass cuts, nail wounds and ulcerations from concomitant ring-worm infections were the common lesions through which the infection entered. The streptococcus was the most common offender, the staphylococcus next in frequency.

Routes of Spread.—These infections spread along fascial spaces or lymphatics, or both. As previously described (Grodinsky, 1929), there are four median plantar spaces which, for convenience, have been designated as M1, M2, M3 and M4, beginning at the surface. M1 lies between the plantar aponeurosis and the flexor digitorum brevis muscle, M2 between the flexor brevis and quadratus plantæ, M3 between the quadratus plantæ and the tarsal and metatarsal bones, and M4 deep to the adductor hallucis obliquis. The interspaces between the slips of the plantar aponeurosis are occupied by sub-

cutaneous fatty tissue (superficial fascia) in the substance of which run the common digital nerves, surrounded by their sheaths derived from the fascial floor of M1, and distally also the plantar metatarsal vessels which become superficial near the necks of the metatarsal bones. Proximally, the digital nerves are in relation to MI but separated from that space by loose, transverse, connective tissue septa about 3 to 4 centimetres proximal to the heads of the metatarsal bones. However, these septa are easily broken through, bringing the superficial portion of the interspaces into direct communication with M1. Deep to the nerves and vessels, and separated from them by a definite and fairly firm layer of fascia, lie the lumbrical muscles surrounded by their thin sheaths derived from those of the flexor digitorum longus and quadratus plantæ muscles, from which they take origin on a line between the middle of the fifth and the head of the first metatarsal bones. The lumbrical sheaths are attached to the lateral walls of the lumbrical tunnels. These walls are formed by the sheaths of the long and short flexor tendons, reinforced by deep bands from the plantar aponeurosis. The potential space between each of the lumbricals (muscles and sheaths) and the overlying layer of fascia is separated from the distal end of M2 by loose areolar tissue which is easily broken through, placing these spaces into communication. Deep to each lumbrical muscle and sheath lies the loose areolar tissue in the corresponding anterior compartment of M₃. This tissue also is easily broken through, thus placing the lumbrical groove into communication with M3. Distally at its insertion into the extensor tendon, each lumbrical extends into the dorsal subcutaneous space.

In addition to the median plantar spaces (M1, M2, M3 and M4), the lumbrical spaces and the dorsal subcutaneous space, there is a dorsal subaponeurotic space deep to the extensor tendons (also a subfascial space between the extensor tendons and overlying deep fascia, as described by Mason and Koch, 1930), a lateral plantar space deep to the abductor digiti quinti and a medial plantar space deep to the abductor hallucis. There is also a medial leg space between the superficial and deep calf muscles brought into relation with the foot by the long flexor tendons behind the medial malleolus, and a lateral leg space deep to the fascial sheaths of the peronei, the tendons of which extend behind the lateral malleolus toward the foot.

The lymph-vessels of the leg follow the superficial and deep veins, the former set being the most important. Those from the lateral side of the foot and ankle accompany the short saphenous vein to the popliteal group of glands, after which they pass through the deep fascia to follow the deep veins. Those from the medial side of the foot and ankle accompany the long saphenous vein to the groin where they enter the subinguinal group of glands and then pass deeply.

The first extension in the present series of cases was usually to the dorsum of the toes and foot. This apparently occurred around the webs through the subcutaneous fascial space (cellulitis), or along the lumbrical spaces when the initial lesion was deep enough to involve the latter. That

this extension was not lymphatic was shown by the fact that there was at this stage no accompanying lymphangiitis or lymphadenitis. At first, the dorsal cellulitis was confined to the region of the toe or toes concerned with the initial lesion, but, with increasing tension, the entire dorsum of the foot became involved. In some cases resolution occurred at this stage—with or without surgical drainage. In others, further extension took place. This was usually up the leg by lymphatic vessels or into the deep plantar spaces by fascial planes. In the latter instances the infection followed the lumbrical muscles from the region of their insertions in the dorsal subcutaneous space or from somewhere along their course through the plantar interspaces towards their origins and into the median plantar spaces M2 or M3, or both. Occasionally, the more superficial median plantar space, M1, was infected directly from the initial lesion by spread proximally along the digital nerves. The spread from the dorsum of the foot proximally up the leg by lymphatics was evidenced by lymphangiitis and lymphadenitis.

In some cases, the primary extension was by lymphatics, but, due to the virulence of the organism or to the poor tissue-resistance of the host, the infection was able to spread beyond the lymph-vessels or glands, producing a cellulitis at some distance from the initial lesion. This was usually in the region of the calf of the leg or the anterior medial aspect of the thigh. As in the hand (Mason and Koch, 1930), an infection of the subcutaneous space of the foot or leg may penetrate through the underlying deep fascia, causing a subfascial or subaponeurotic infection, or even bone-and-joint involvement.

Diagnosis.—This consists chiefly in the interpretation of the physical signs in terms of anatomic structures so that the initial location of the infection and the routes of spread may be accurately determined and even anticipated. The depth and character of the lesion through which the infection enters will give some hint as to the space first involved. The location of the swelling, redness and localized tenderness in relation to the course of the lumbrical muscles would indicate whether or not the spaces about the latter were serving as pathways for the spread of the infection. On the other hand, swelling, redness and tenderness at the web on the opposite side of the toes (lateral side) or all around the toes would indicate a spread by the subcutaneous fascial space. Lymphatic involvement is easily determined by the red streaks up the leg and enlarged, tender lymph-glands (lymphangiitis and lymphadenitis) in the proper anatomic location. Involvement of the deep median plantar spaces (M1, M2, M3) is indicated by localized tenderness, deep, brawny induration and change in contour of the foot-especially the obliteration of the concavity of the longitudinal arch. Secondary involvement of the dorsal subfascial and subaponeurotic spaces probably requires exploratory incision through the overlying cellulitis before the diagnosis can be made with certainty. The X-ray will help in diagnosing bone-and-joint lesions.

Treatment.—The first essential for successful treatment is an accurate diagnosis of the type and anatomic location of the infection as well as a

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knowledge of the common routes of extension. The dictum laid down by Kanavel (1921) and others, that infections due to the streptococcus should never be incised until localization has occurred and lymphangiitis has subsided, holds true in the leg as well as in other regions. Rest and moist heat (hot packs) are the best means of treatment until localization takes place. Once the latter has been obtained, long incisions accurately placed, with or without rubber tissue drainage, should be made. The location of these incisions will depend upon the degree of spread of the infection. If still confined about the primary lesion, simple enlargement of this opening may be sufficient. If localization has occurred over the dorsum of the proximal phalanx and the adjacent part of the foot, an incision should be made over the area of the greatest softening or induration, or on both sides of the toes, after which it is usually necessary to connect these with the plantar wound by undermining or continuous incision around the web. When infected, lumbrical spaces should be opened from the insertions of the muscles on the dorsum, around the medial side of the corresponding toe, and through the web to the plantar surface as far as the ball of the foot.

As suggested in a previous study (Grodinsky, 1929), the best way to open the deep plantar spaces (M1, M2 and M3) is by a medial incision along the plantar border of the first metatarsal bone, passing between the bone and the muscles of the great toe to reach the medial wall of these spaces and then plunging through this wall at the proper level with a hæmostat to reach the space or spaces involved.

REPORT OF CASES

CASE I.-L. G., male, aged seven. University Hospital, No. 29064. About July 1, 1929, the patient "skinned" the dorsum of his left foot near the second metatarsalphalangeal joint. He first complained of pain July 5 and this was located in the inguinal region. Two days later the dorsum of the foot became red, swollen and painful. Upon admission to the hospital the following day (July 8, 1929), he had a rectal temperature of 100.6° and a pulse of 108. Local examination revealed a marked swelling and redness over the entire dorsum of the foot but centring about the region of the second metatarsalphalangeal joint, where deep induration (fluctuation) and localized tenderness could be distinguished. The swelling extended into the dorsum of the second toe but the other toes were not involved. Proximally, it extended to the annular ligament, and transversely between the first and fifth metatarsal bones. The left subinguinal lymph-glands were enlarged and tender but not fluctuating. There was no lymphangiitis. There were a little swelling and redness on the plantar side of the web between the great and second toes, but closer to the latter. The white blood-count was 10,900 with 82 per cent. polymorphonuclears. The blood sedimentation time was thirty minutes to the 18 millimetre mark (Linzenmeier technic).

Hot, moist, boric-acid packs were applied from the toes to the groin for forty-eight hours, after which, under nitrous oxide-oxygen anæsthesia, incisions were made on both sides of the dorsum of the proximal phalanx of the second toe and carried proximally into the foot about 4 centimetres. Approximately 3 drams of thick yellow pus were evacuated from the subcutaneous space and through-and-through rubber tissue drainage between the incisions instituted. A hæmostat was passed around the web on the medial side of the second toe to the plantar surface where a counter incision was made and through-and-through drainage established between dorsal and plantar incisions. A hot,

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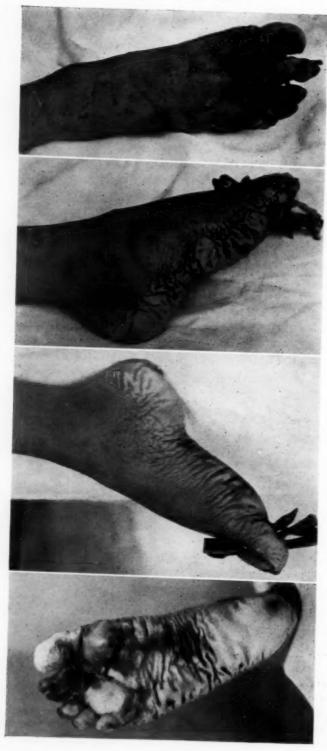


Fig. 1,a,b,c,d,-Case II. Photographs taken just before the second operation, showing fulness over the median plantar surface, and obliteration of the concavity

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wet dressing was applied. Culture from the wound showed staphylococcus aureus. After this operation, the patient made an uneventful recovery and was dismissed from the hospital with all wounds healed on July 27, 1929, seventeen days after drainage was instituted.

Summary.—The infection no doubt entered through the abrasion on the dorsum over the second metatarsal-phalangeal joint. First extension by lymphatics to the subinguinal group of glands. Later, a subcutaneous collection which spread to involve the entire dorsum of the foot but centred about the original portal of entry. A beginning extension along the first lumbrical muscle toward the plantar side but no involvement of the deep plantar spaces. Proper drainage of the spaces infected led to an early resolution.

CASE II.—D. S., male, aged six. University Hospital, No. 32277. June 28, 1930, the patient cut the plantar surface of his right second toe on a piece of glass. A few days later (July 1, 1930) the sides of the toe, particularly the medial side, became swollen, red and tender. From there the infection spread to the dorsum of the foot which became red and swollen over the distal one-half, centring about the second toe. Upon admission to the hospital (July 2, 1930), the patient had a rectal temperature of 102°, pulse 120, respirations 22. Local examination showed a ragged laceration on the plantar side of the middle phalanx of the right second toe. There were swelling and redness at the sides, especially the medial side, extending to the dorsum of the toe and the adjacent part of the foot. No definite fluctuation or deep induration. A white blood-count at this time was 16,000 with 70 per cent. polymorphonuclears. Continuous hot boric-acid packs were applied.

First operation (July 11, 1930).—There were now definite induration and deep fluctuation over the medial side and dorsum of the second toe, and pus was draining from the original laceration. There was some swelling and tenderness over the ball of the foot proximal to the second toe. Under ether anæsthesia, the edges of the lacerated wound were trimmed away allowing a great deal of pus to escape. By probing and pressure the source of the pus was located at the sides and dorsum of the toe, and proximally along the flexor tendons towards the ball of the foot. The tendon sheath was apparently not involved, the infection passing superficially and especially medially along the lumbrical groove. An incision was made on each side of the dorsum of the toe and connected with the original wound by through-and-through rubber tissue drains. An incision was made over the infected lumbrical space from the plantar surface of the web to the ball of the foot. There seemed to be no involvement proximal to this point at this time. Culture from the wounds showed hæmolytic streptococcus. After this operation the temperature varied from 98° to 102°, pulse 90 to 120. With the increasing and persistent temperature, a definite fulness became evident over the median plantar surface of the foot, extending as far back as the base of the fifth metatarsal bone. Transversely, this fulness extended between the lateral side of the first and medial side of the fifth metatarsal bones, obliterating the concavity of the long plantar arch. (Fig. 1,a,b,c,d.) There was definite tenderness over this area and pressure caused the appearance of pus at the plantar incision previously made.

Second operation (July 21, 1930).—Under ether anæsthesia, an incision was made on the medial side of the foot opposite the plantar surface of the first metatarsal bone and extending the length of that bone. The abductor hallucis, the flexor hallucis brevis and the superimposed longus tendon were elevated and a hæmostat passed between them and the metatarsal bone into the median plantar space containing a probe inserted through the old plantar wound. The latter was now lengthened proximally almost to the middle of the foot, the incision passing through skin, superficial fascia, plantar aponeurosis and between two slips of the flexor digitorum brevis. The space exposed by both medial and plantar incisions proved to be M2, lying between the flexor digitorum brevis and quadratus plantæ muscles. It was a definite, localized space similar to that demonstrated in the dissecting room (Grodinsky, 1929). Through-and-through drainage was established and hot, wet dressings applied.

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The patient recovered rapidly after the second operation. (Fig. 2,a and b.) The temperature became normal on the seventh post-operative day and remained normal after that. The drainage rapidly subsided and healing was complete at the time of his dismissal from the hospital, twenty-three days after this operation. When seen in the follow-up clinic one month later, the foot appeared normal except for the plantar, dorsal and medial scars. (Fig. 3,a,b,c.) The patient walked with a slight limp, probably due to the irritation from the plantar scar which might have been avoided by making only the medial incision for the drainage of the deep plantar space M2.

Summary.—The infection entered the lacerated wound which extended to but not through the fibrous sheath of the flexor tendons of the second toe. Spread subcutaneously and along the first lumbrical muscle to the sides and dorsum of the second toe and the dorsum of the foot. Then spread proximally along the first lumbrical muscle (and superficial to it) into the median plantar space M2. Possibly some extension



FIG. 2,a,b.—Case II. Photographs showing condition of the foot ten days after the second operation, and location of the incisions for drainage of the median plantar space, M2.

proximally superficial to the flexor brevis tendon of the second toe but no definite evidence of pus in MI or in any of the other median plantar spaces except M2.

Case III.—J. G., female, aged fifty-one. University Hospital, No. 33196. September 26, 1930, the patient stepped on a rusty nail which entered the ball of the left foot in line with the great toe. A few days later there was evidence of infection in the wound and her physician enlarged it, obtaining some thick pus. Hot, moist dressings were applied but the infection continued to progress and spread to the dorsum of the foot, which became red and swollen. At this time (October 6, 1930), there was also evidence of lymphatic extension up the medial side of the leg and thigh. Her physician made an additional incision over the dorsum of the proximal phalanx of the great toe. On admission to the hospital (October 7, 1930), the patient showed marked constitutional symptoms: temperature 104° to 106°, pulse 100 to 130, respirations 20 to 50. Her mentality was not clear enough to answer questions. She was in a restless type of delirium which

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soon passed into coma. Local examination showed thick pus exuding from a small incision over the plantar aspect of the first metatarsal-phalangeal joint. The ball of the foot was greatly swollen but particularly so in the medial half. There was no median plantar space involvement. There was a definite cellulitis over the dorsum of the foot centring about the first and second toes but extending laterally to the fifth metatarsal bone, proximally to the bend of the ankle and distally to the webs. There were redness, swelling and induration but no fluctuation. No secretion was coming from the small incision over the dorsum of the proximal phalanx of the great toe. There was a wide strip of lymphangiitis extending along the course of the internal saphenous vein to the mid-thigh region. There were a few palpable subinguinal lymph-glands. A white blood-count on admission was 10,500 with 96 per cent. polymorphonuclears. The next morning it had risen to 16,000 with the following Schilling count: segmented, 27 per cent.; staff, 35 per cent.; young, 26 per cent.; myelocytes, 3 per cent.; myeloblast, 1 per cent.;



Fig. 3,a,b,c.—Case II. Photographs showing condition of the foot with plantar, medial and dorsal scars seven weeks after the second operation.

lymphocytes, 8 per cent. There was a very rapid blood sedimentation rate, seventeen minutes to the 18 millimetre mark. Culture from the wounds showed staphylococcus aureus. A blood culture showed a pure growth of staphylococcus aureus. The urine contained albumen 3 plus and granular casts 2 plus.

On admission, the patient was given general supportive treatment and continuous hot, moist, boric-acid packs locally. The next morning the plantar incision was enlarged freely and carried deeply to the long and short flexors of the great toe and the first lumbrical groove laterally. A hæmostat was carried superficially and deeply through the first web to the incision on the dorsum of the great toe which was enlarged the extent of the undermined dorsal subcutaneous space (about 3 inches long). Through-and-through rubber tissue drainage was established. (Fig. 4a,b,c.) The patient went into deeper coma, became cyanotic and her respirations became very rapid (65). There was some evidence of lobar pneumonia in the right base. The temperature hovered around 106°, pulse 130. She died during the night and no autopsy was granted.

Summary.—Subcutaneous infection of ball of foot in line with great toe. Spread subcutaneously and along the first lumbrical muscle to the dorsum of the first and second toes and the dorsum of the foot. Extension through the deep fascia from the dorsal



Fig. 4.a.b.c.—Case III. Photographs showing plantar, medial and dorsal views of the foot and leg shortly after drainage was established. Note the leg and the blebs just above the knee,

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subcutaneous to the dorsal subaponeurotic space in the region of the first and second toes. Lymphatic spread from the dorsum of the foot along the channels accompanying the internal saphenous vein—lymphangiitis and lymphadenitis. Septicæmia. Terminal right lobar pneumonia. Death.

Case IV.—H. S., male, aged sixty-five. University Hospital, No. 29179. About two weeks before admission to the hospital, the dorsum of his left foot opposite the second and third toes and the proximal phalanx of the latter became red, swollen and painful. This condition persisted and was present on his admission to the hospital (July 23, 1929). At this time his temperature was 99.8°, pulse 84, respirations 22. There were no open wounds except an ulcerating soft corn between the second and third toes. X-ray examination showed an osteomyelitis of proximal phalanx of the third toe and adjacent part of the foot yielded thick yellow pus. The wound continued to drain until a sequestrum of bone came away spontaneously from the proximal phalanx of the third toe (August 6, 1930). After that, the wound healed rapidly.

Summary.—Subcutaneous infection in web between second and third toes. Spread subcutaneously (possibly also along first lumbrical muscle) to the dorsum of the proximal phalanx of the third toe and the dorsum of the adjacent part of the foot. Sloughing of the extensor tendons of the third toe with extension of the infection into the dorsal subaponeurotic space over the first phalanx of the third toe. Spread to the bone with osteomyelitis and necrosis resulting.

CASE V.-J. W., male, aged twenty-three. University Hospital, No. 20042. July 4, 1929, the patient first noticed pain, swelling and redness of the third toe of the left foot with red streaks appearing on the dorsum of the foot. There was no history of injury but the patient suffered from ring-worm infection of the toes (athletic foot) and had scratched some of the latter lesions on the plantar surface of the third toe. Upon admission to the hospital (July 15, 1929), the third toe was about twice normal size, and there were redness and induration over the dorsum of the proximal phalanx. The dorsum of the foot was swollen but not indurated or fluctuating. Red streaks (lymphangiitis) passed up the dorsum of the foot behind the lateral malleolus to the back of the leg where they extended as far as the popliteal fossa. The popliteal lymph-glands were palpable. The temperature varied from 99° to 103°, pulse 70 to 120, respirations 18 to 27. The white blood-count was 12,200 with 86 per cent, polymorphonuclears. Hot, moist packs were applied until July 9, 1930 when there seemed to be sufficient localization and fluctuation over the dorsum of the proximal phalanx of the third toe to warrant incision of this area. This was done and free pus obtained from the subcutaneous space. A culture from the wound showed hæmolytic streptococcus. The temperature came down to 100° for the next two days after which it again rose to 102°. Meanwhile, the calf of the leg became red, swollen and indurated, and it became evident that localization was occurring in this region. A blood culture was negative after three and five days. The blood sedimentation test was twenty-three minutes for 18 millimetres. White blood cells, 15,400. Schilling: segmented, 73 per cent.; staff, 12 per cent.; young, 2 per cent.; large lymphocytes, 7 per cent.; small lymphocytes, 2 per cent.; mononuclears, 4 per cent.

July 13. 1929, under gas-oxygen anæsthesia, a long incision (10 inches) was made over the calf of the leg from the middle of the popliteal fossa to the middle of the leg. The skin and subcutaneous tissues were divided and free pus found in the latter layer, which was ædematous and bled very little. At the upper third of the leg there was an erosion through the deep fascia through which free pus escaped. This opening was enlarged and the pus found to come from the substance of the gastrocnemius muscle and the space between that muscle and its sheath. A rubber tissue drain was laid lengthwise in the wound which was left wide open, and a hot boric-acid dressing applied. A culture from the wound showed hæmolytic streptococcus. After this operation, the condition of the patient improved rapidly, the temperature reaching normal on the fifth

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post-operative day and remaining normal after that. The drainage, which was free at first, gradually subsided. The margins of the wound were approximated with adhesive plaster and the patient was dismissed from the hospital August 3, 1929 with the wound completely healed, almost by primary union.

Summary.—The portal of entry was no doubt one of the ring-worm lesions on the plantar side of the third toe. Secondary infection with the streptococcus by scratching. A subcutaneous spread around the toe to the dorsum where localization occurred over the proximal phalanx. Lymphatic extension from the dorsum of the toe and foot through the vessels accompanying the short saphenous vein to the popliteal fossa, where enlarged glands could be felt. Further extension through the walls of the lymph-vessels in the calf of the leg, forming an extensive cellulitis in that region. Finally, an erosion through the deep fascia into the space between the gastrocnemius muscle and its sheath, and into the substance of the muscle itself.

CASE VI.—E. C., male, aged twenty-seven. University Hospital, No. 29216. July 15, 1929, the patient cut the plantar surface of his right second and third toes on a piece of tin. He paid little attention to this wound and it healed rapidly. July 22, he noticed a lump appearing high up on the anterior medial aspect of the right thigh. This lump progressively increased in size and became more painful until his admission to the hospital, July 27, 1929. At this time his temperature was 101.4°, pulse 104, respirations 24. White blood cells 16,900. Schilling: segmented, 34 per cent.; staff, 44 per cent.; young, 2 per cent.; large lymphocytes, 15 per cent.; small lymphocytes, 3 per cent.; mononuclears, 2 per cent. Blood sedimentation test: thirty minutes to 18 millimetre mark. Locally, there was a large, red, indurated, fluctuating area over the anterior medial aspect of the right thigh from the inguinal ligament to the junction of the superior and middle thirds of the thigh. It looked like a cellulitis which had become localized and softened in the centre. Enlarged subinguinal glands were palpable in the upper part of the area involved. The patient was given a nitrous oxide-oxygen anæsthetic and a generous incision made vertically through the region of greatest localization. A counter incision was made higher up parallel and just distal to the inguinal ligament. Throughand-through drainage was established with rubber tissue and hot, moist dressings applied. Pus from the wounds showed streptococcus hæmolyticus. The temperature dropped by lysis, reaching normal on the fifth post-operative day. The wound, after draining freely for several days, gradually closed and the surrounding inflammatory reaction subsided. After his dismissal from the hospital (August 13, 1929), there were several local recurrences and residual abscesses which were opened and drained. Complete healing was finally obtained about one month after his dismissal from the hospital.

Summary.—A streptococcus hæmolyticus infection; starting in the wound on the plantar aspect of the right second and third toes, spreading from there along the lymphatic vessels accompanying the long saphenous vein to the subinguinal group of lymph-glands, and resulting in an acute adenitis and periadenitis. Extension from the infected lymph-vessels and glands to the surrounding subcutaneous tissue in the form of a cellulitis which finally localized on the anterior medial aspect of the upper third of the thigh. Several residual abscesses, due to the persistence of the infection within the subcutaneous space after healing had apparently been complete.

SUMMARY AND CONCLUSIONS

A study of a group of foot cases which comprise a clinical entity in that they have a common starting place and, in general, similar routes of spread—the differences being chiefly those of degree of extension before resolution begins or death occurs. The portal of entry is through lesions about the toes, usually on the plantar surface. The common infective organisms are the streptococcus hæmolyticus and staphylococcus aureus. The routes of

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spread are the fascial spaces, described in a previous study (Grodinsky, 1929), and the lymphatic channels accompanying the long or short saphenous veins. The diagnosis consists mainly in an interpretation of the physical signs in terms of anatomic structures so that routes of spread may be accurately determined and anticipated. The treatment of lymphatic infections is conservative until localization has occurred. Fascial space infections are drained as soon as the diagnosis is made, the efficiency of the drainage depending upon the accuracy of the anatomic diagnosis and the location of the incisions. Six representative cases are reported in detail.

BIBLIOGRAPHY

- Grodinsky, Manuel: A Study of the Fascial Spaces of the Foot and Their Bearing on Infections. Surg., Gynec. and Obst., vol. xlix, pp. 737-752, 1929.
- Grodinsky, Manuel: A Study of the Tendon Sheaths of the Foot and Their Relation to Infection. Surg., Gynec. and Obst., vol. li, pp. 460-468, 1930.
- Kanavel, Allen B.: Infections of the Hand. Fourth Edition, Lea & Febiger, Philadelphia and New York, 1921.
- Mason, Michael L., and Koch, Sumner L: Human Bite Infections of the Hand; with a Study of the Routes of Extension of Infection from the Dorsum of the Hand. Surg., Gynec. and Obst., vol. li, pp. 591-626, 1930.

TRANSACTIONS

OF THE

NEW YORK SURGICAL SOCIETY

STATED MEETING HELD FEBRUARY 25, 1931

The President, Dr. John Douglas, in the Chair

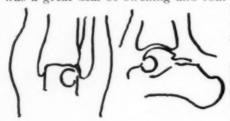
OSTEITIS CYSTICA FIBROSA OF ASTRAGALUS AND XANTHOMA OF DORSUM OF FOOT

Dr. CLAY RAY MURRAY presented a man, aged thirty-six, who was admitted to the Presbyterian Hospital May 2, 1930, with a history that twenty years before he had sustained a severe sprain of the right ankle. The swelling has never completely subsided, and while the function of the foot has been a little impaired, and there has been occasional slight pain, the condition has remained stationary and has never kept him from work.

Seven hours before admission, while at work, he turned his right ankle. Although he had considerable pain, he continued to work. In one hour there was a great deal of swelling and four hours later the pain was so great that

he could not walk on the foot. Maximum pain around internal malleolus and up anterior aspect of the leg.

When admitted there was diffuse and brawny swelling of the ankle and dorsum of the foot, most pronounced over the internal malleolus. This was very great, and the foot was twice its normal size. There was an impression Fig. 1.—Location of eavity in astragalus of definite fluctuation and the feetwith break in cortex, indicating amount of soft ing of rather small nodular bodies in the deep tissues.



Attempted aspiration of the joint cavity yielded only 2 cubic centimetres of blood-tinged fluid. This was subsequently negative for organisms. He had a temperature of 100.2° by mouth. An X-ray showed a bone cavity in the astragalus, apparently broken through at one point near the joint margin, the tremendous swelling of the soft parts, and a mottling of them, with the presence of what seemed to be gas bubbles. (Fig. 1.) The soft parts were hot and extremely tender, the diffuse nodular feel of the deep tissues suggested the possibility of crepitus, and a white-cell count showed 11,700 cells with 78 per cent. of polymorphonuclears.

The ankle was explored the following morning by Doctor Schullinger. He noted unusual vascularity of the soft parts; and their unusual appearance. The tissues beneath the skin were yellow in color, soft and friable, brownishred in places, somewhat ædematous and elastic to the feel. There were but 2 cubic centimetres of pinkish fluid in the joint. The tissue described surrounded the tendons, and villi of the same nature lay within the joint cavity. There were no areas of caseation or necrosis. Exploration of the astragalus close to the fibula failed to find any definite cavity. As much of the peculiar tissue described was removed as could be taken away, but practically the whole ankle and dorsum seemed involved. The tendons were not diseased. The wound was packed with iodoform gauze after aërobic and anaërobic

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cultures for injection into guinea pigs were taken. These subsequently proved negative. The microscopic report of the tissue removed follows:

Gross.—Specimens are a number of fragments of amorphous tissue, said to be curettings from the ankle-joint. They are relatively soft, pale and some of them are stained different tints of red and yellow.

Microscopic.—Some sections have been taken through the lining of the ankle-joint. They show that the synovial and subsynovial tissue is enormously thickened and thrown into villous folds. There has been a marked proliferation of thick-walled capillaries and about these an astonishing growth of fat, ovate cells. It is due chiefly to these stroma cells that the bulk of the tissue has been so materially increased. The cytoplasm of these cells has undergone various types of degeneration. In places it is apparently of a mucinous type; in others, the cells are of the foam variety due to accumulation of lipoids in tiny vacuoles. It is due to groups of these cells that the tissue grossly stained yellow. In other areas, brown blood pigment is found in the cytoplasm of some of these cells. Aside from the phagocytic degenerative activity, they show no evidence of rapid or unnatural division. There is no evidence of tuberculosis. The condition is apparently not a neoplasm but an inflammatory reaction. Diagnosis.—Villous arthritis of ankle-joint.

For the next month the condition remained unchanged with a large, pouting, granulating cavity. X-ray still showed the cavity in the astragalus.

On June 9, the ankle was re-explored. At this time the findings were practically identical with the preceding operation, except that the cavity in the astragalus was located and opened up and the rather normal-looking granulation tissue filling it was removed. Anaërobic and aërobic cultures from tissue and bone were taken, all of which proved negative on culture and guinea pig. A counter opening was made on the inner side of the foot, and through-and-through packing established.

The pathologic report follows:

Gross.—Specimen consists of numerous irregular pieces of tissue, the largest measuring 3 by 2 centimetres. The first specimen consists of a piece of skin in the centre of which is a sinus running the depth of the specimen. Apart from the presence of the sinus opening, this specimen shows nothing remarkable. There are also four pieces of tissue, the largest about 2 centimetres in length, said to come from the neighborhood of the joint capsule. On section they show fibrous tissue in the midst of which are a few small yellowish specks, which, when cut, do not grate like calcium. Similar tissue, taken from the neighborhood of the tendon sheath, shows on section a white appearance with yellow centre, which may be necrosis. There are other specimens containing bone and cartilage from the astragalus and astragalo-tibial joint. The cartilage has lost its smooth, regular appearance. The bone comes from the margin of a cavity which contains soft, peculiar-looking material, which is yellow in color when sectioned.

Microscopic.—One section was taken through cartilage and bone, including the margin of the bone cavity and the soft tissue which lay in it. It shows erosion of the cartilage. The bone beneath the cartilage is composed of irregular shapes, among which lies vascular fibrous tissue. Only in one small portion is there evidence of increase of the number of cells in this area. The soft tissue in the bone cavity is fibrous, not very cellular and contains frequent capillaries. A number of large multinucleated cells are seen near the bone margin. Another section taken from the tissue within the cavity shows much the picture described above.

Section from the soft tissue near the joint cavity shows granulation tissue thrown up into irregular folds resembling villi. Parts of this specimen appear to be undergoing degeneration. Section from the region of the tendon sheath shows masses of cells closely packed together, resting upon a varying amount of fibrous structure. The cells are of almost uniform size, polygonal in shape, with granular cytoplasm and oval nuclei which stain deeply and usually show a definite nucleolus. These cells apparently grade off into collections of other cells whose cytoplasm is foamy, resembling xanthoma cells. It is

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possible to find many instances of gradations between the two types of cells described. With the Laidlaw stain the foamy cells do not take the silver stain but the other cells do. This suggests that they are not of connective-tissue origin. In one portion of the specimen is a mass of pink-staining material somewhat suggesting bone in contour but showing no calcification. In all the specimens much yellow pigment, thought to be blood pigment, is present. *Diagnosis*.—Xanthoma of tendon sheath; osteitis fibrosa cystica of astragalus; villous arthritis of astragalo-tibial joint.

This specimen was shown to Dr. James Ewing, who suggested the above diagnoses, He recommended radiotherapy and believed that amputation was contra-indicated. He thought that with radiotherapy the prognosis was good.

The patient was accordingly given progressive and intensive radiotherapy, with marked improvement at first, followed by increased swelling and pain later. He was seen again by Doctor Ewing, who felt that the diagnosis of xanthoma was correct, and that the patient's symptoms were due to overradiation. Accordingly, all radiotherapy was stopped, and rest alone was given to the foot. Under this routine his wounds healed up, and functional treatment by physiotherapy was instituted.

In the intervals between treatment periods he had an inguinal hernia operation. Seen a third time by Doctor Ewing, after he had again studied the data and sections and expressed some doubt as to the diagnosis, the final opinion expressed was that the soft tissue mass was a xanthoma, that it would not metastasize but would recur locally after surgery. He advised continuation of radiotherapy with amputation as a final resort.

His condition today is very much improved, but there are still swelling, pain and disability, and he is certainly not cured.

The case is presented because of the unusual pathologic picture, the fact that because of the trauma which preceded the acute and incapacitating symptomatology the patient is being awarded compensation and because of the uncertainty of future prognosis, and of future procedure.

VOLKMANN'S ISCHÆMIA FOLLOWING SUPRACONDYLAR FRACTURE OF HUMERUS

Doctor Murray presented a girl, nine years of age, who was admitted to the Presbyterian Hospital, November 16, 1930, for treatment of Volkmann's ischæmia following a supracondylar fracture of humerus. The fracture had been sustained November 4, 1930, and was at first put up in an acutely flexed position. Some hours later there followed great pain and distress and marked swelling of the whole elbow, forearm and hand. The hand was bluish in color and cold. The elbow was taken out of the Jones' position and splinted with posterior basswood in extension. This somewhat relieved the child's pain. An X-ray the next morning showed a supracondylar fracture with complete posterior and marked outward displacement of the lower fragment. The radial pulse at this time could not be appreciated, the hand was blue and sensation was absent from the fingers and no finger motion and very little wrist motion were possible. The arm was kept in elevation with heat and in extension in a splint. The next day the skin from elbow to wrist showed a great number of large blebs. The treatment by rest, elevation and heat was continued in an attempt to get rid of swelling before attempting reduction. On the thirteenth day, when seen by Doctor Murray, there was still marked swelling; the blebs were present although largely dried up; there was no perceptible radial pulse; the hand was blue and cold; wrist extension was impossible; finger motion was completely lost, and there was no sensation in fingers or hand. Her X-rays at this time showed profuse callus formation with the position shown in Fig. 2. The bony deformity could be readily diagnosed clinically. At the hospital a closed attempt at reduction was made. It proved impossible to move the

fragments and a Kirschner pin was therefore driven by motor through the olecranon at the elbow, and the arm placed in overhead traction with over 20 pounds' pull. (Fig. 3.) Operative procedure was rendered dangerous by the condition of the skin. Despite the callus, some improvement was secured by the pin traction. Intensive physiotherapy in the form of heat, light massage and muscle stimulation was applied and in twenty-four hours a radial pulse was palpable. The traction was maintained for some days while physiotherapy was continued. By that time practically all swelling was removed from the part, some wrist motion was present, the color of the hand was distinctly nearer normal and a slight return of sensation was apparent. The blebs were clearing up rapidly but no finger motion was present and beginning contracture of the flexor tendons was becoming apparent. She was fitted with an adjustable cock-up for both fingers and wrist to minimize the degree and rapidity of tendon contracture and sent home to have physiotherapy of the type described as well as galvanism and

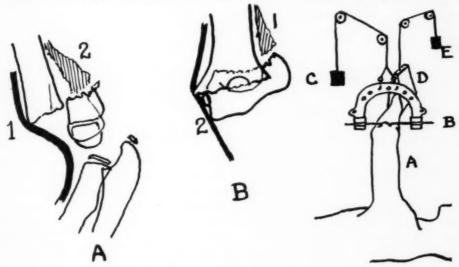


Fig. 2.—Supracondylar deformity at thirteen days. A—Lateral view. Note new bone at 2 and the distortion of brachial vessels and median nerve at 1 which is responsible for circulatory interference and swelling. The effect of acute flexion without reduction is obvious as regards the vessels.

Fig. 3.—Kirschner pin traction in supracondylar fracture. A —Upper arm in vertical traction by B pin through olecranon. D— Hand suspended with forearm horizontal by weight E. C is 20 pounds traction weight.

faradism. It is now four months and the process has reached its maximum. It is intended now to remove the projecting lower end of the upper fragment and to transplant the epicondyle with the flexor group attachments down the ulnar shaft a sufficient distance to allow of hyperextension of the fingers and to then work for further functional return.

The case is cited to call attention to the rapidity of development of Volkmann's ischæmia, to the common misunderstanding of the Jones position as a method of reduction instead of a position of immobilization, and the absolute necessity for early reduction regardless of the amount of swelling, since it is the bony deformity which is responsible for the circulatory interference. These cases are frequently very difficult to reduce, particularly with the rapid marked swelling which occurs, and frequently require considerable

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violence in the reduction. The reduction may be difficult to maintain because of the marked soft part swelling. The Kirschner pin traction is a valuable adjunct in securing early rapid reduction in these cases with practically no trauma to the site of fracture, leaving the whole arm, elbow, forearm and hand free for early intensive physiotherapy. In two cases recently at the hospital, almost identical in deformity with this one, practically anatomic reduction was secured after closed reduction had failed. In the first case, a fresh one, anatomic reduction could be secured by closed methods, but the position could not be maintained. In the other, four days old when first seen by Doctor Darrach, he could not secure satisfactory position by closed reduction. Both cases showed signs of threatening Volkmann's ischæmia. In the early case anatomic reduction was secured with the Kirschner pin traction in a few hours. In Doctor Darrach's four-day-old case anatomic reduction was secured in between forty-eight and seventy-two hours.

COMPRESSION FRACTURE UPPER DORSAL SPINE

Doctor Murray presented a man, aged twenty-seven, who was admitted to the Vanderbilt Clinic December 8, 1930, complaining of pain in the middle of the upper back since November 19, 1930, on which day while diving in unexpectedly shallow water in Miami, he struck his head a glancing blow. He noted pain between his shoulders immediately. At first the pain was constant, later it gradually subsided, and at present there is pain only on certain motions—rotation of head especially.

Physical examination was negative except for the back. This showed a slight palpable prominence of the sixth dorsal spine, a little diffuse tenderness from the fifth to the seventh dorsal, most marked just below the sixth spine and some muscle spasm in the paravertebral muscles from the fifth to the seventh segments. The pain was slight and only on sudden turning motions

of the head and shoulders.

An X-ray examination showed in the lateral view a slight though definite decrease in the vertical diameter of the seventh segment with slight buckling along its anterior margin, a compression fracture of the seventh dorsal.

The case demonstrated the sparsity of clinical signs in typical compression fractures and emphasized the frequency of the diagnosis of muscle bruise, muscle strain, sprained back, lumbago, myositis and neuritis. The violence being always indirect—a "jack-knifing" mechanism—the history, with absence of direct violence to the injured site, lends itself to these diagnoses readily.

As to the treatment indicated in these cases, in this particular case, the deformity being exceedingly slight and the injury three weeks old, the patient was fitted with a Taylor spinal brace, remaining in bed at home until the brace was ready. He was instructed to wear the brace twenty-four hours a day for at least three months, following which time, provided everything was satisfactory, it could be removed at night when the patient was in bed but must be worn all day for at least another three months. So long as he wears the brace he is comfortable. The last X-ray was taken on the second

of this month and shows no increase in deformity and no absorption in the vertebral body.

Doctor Murray said that his general concept of the treatment of these cases is that it should follow the general principles of treatment for all fractures. In fresh cases they therefore attempt by hyperextension to correct the deformity by the method of Rogers, of Boston, or Davis, of Erie, Pennsylvania. If they are unable to correct the deformity they advise fusion of the Hibbs type. If the deformity can be corrected they should be immobilized in bed in a hyperextension plaster jacket for eight weeks, X-raying through windows cut in each side of the jacket. If the correction cannot be maintained or if there is evidence of absorption in the vertebra, as shown by weekly X-rays at any time in this period, fusion is advised. After eight weeks of plaster a spinal brace is fitted in hyperextension and the patient wears this for at least six months—three months day and night and three months daytime only. This period is prolonged if the vertebra does not show normal density. If at any time in this period recompression is seen to be occurring, or absorption of the body, as evidenced by monthly X-rays, fusion is advised. Finally, if after apparently perfect healing has occurred there persist pain and spasm in the back—which may be due to some malalignment or "strain" in the articulations not visible by X-ray-fusion is advised. In all cases seen late—a month or more after injury—with deformity, correction cannot be hoped for and fusion is advised. If refused, the same routine as here advised is carried out without the preliminary correction.

In fresh cases there is no reason for fusion without preliminary attempted correction of deformity. In cases which can be corrected fusion is not justified in average hands unless the correction cannot be maintained, or absorption occurs, or pain symptoms persist despite healed correction.

In cases which can be corrected less than 15 per cent. require fusion on these grounds if properly treated. Primary fusions fail in about 15 per cent. of the cases. The disability time in the 85 per cent. who heal successfully under either method of treatment, in the fresh cases which can be corrected, is approximately the same.

MALUNION COLLES FRACTURE; MEDIAN NERVE PALSY; TENOSY-NOVITIS

Doctor Murray presented a man, aged thirty-six, who was admitted to the Presbyterian Hospital September 6, 1930, with a diagnosis of "Faulty Union Following Colles's Fracture." Three months before admission, he fell on the outstretched right hand. He was taken to a local hospital and three days later "when the swelling reduced" the ankle and the wrist were "put in plaster." This circular plaster was left on for five weeks. Following this, physiotherapy and active motion were instituted. On admission, patient complained of deformity of wrist, numbness and coldness of index, middle and ring fingers, loss of motion in wrist and fingers and loss of strength in the hand. Examination showed obvious deformity, almost complete median nerve paralysis and stiffness of fingers due apparently to tenosynovitis of the flexor tendons with marked limitation of wrist movements in all directions.

X-rays revealed marked posterior displacement and tilting backward of the lower radial fragment, marked radial displacement and a projecting sharp anterior edge of the lower end of radial shaft over which the median nerve and tendon sheaths were stretched. Operation to correct this deformity

was done September 8, 1930.

The operative procedure was rendered difficult by the fact that the fracture line was very low in the radius with a resultant small lower fragment and that the original impaction had destroyed a certain amount of bone making it impossible to restore the full length of the radius and thereby correct the marked radial deviation of the hand. The wound healed by primary union and four days later beginning return of finger sensation was noted, and also increasing freedom of finger motion. The wrist was up in palmar flexion and as much ulnar deviation as could be secured. On the twenty-third day. it was noted that finger motion was almost complete and that sensation was rapidly returning to fingers. On the twenty-sixth day, his splints were removed and active use of the wrist and physiotherapy started (diathermy and static brush before and after exercise). On the thirty-second day, range of motion in wrist was from 150° to 180°. At fifty-three days, there were present two-thirds of normal supination and pronation with wrist flexion and extension range from 140° to 190°, practically complete finger motion and only slight traces of nerve involvement. He was re-admitted to the hospital November 18, 1930, for subperiosteal resection of the ulnar head to increase pronation and supination range and to diminish the remaining deformity, if possible. Doctor Darrach resected the ulnar head subperiosteally November 21, 1930. His wound healed by primary union. No splints were applied and active motion was started within three days. On his discharge November 26, 1930, it was noted that he could flex, extend and supinate more than before operation five days previously.

December 17, 1930, six months after original injury, three months after the first operation, extension was to 210°, flexion to 120°, pronation 7/8, supination 4/5. Today, five and one-half months after the first operation, he has a little better than that. The strength of grip is considerable and is constantly increasing. The nerve symptoms have practically completely disappeared and he has full finger motion. The æsthetic result is not good—with the original deformity and actual loss of bone it could hardly be so.

But it makes a useful wrist out of a hopelessly crippled one.

LATE REMOVAL OF SCAPHOID FOR UNUNITED FRACTURE

Doctor Murray presented a man, aged thirty, who was admitted to The Vanderbilt Clinic August 9, 1929, with history of having been hurt by kick-back of a car he was cranking, with resultant pain and disability in the wrist. The accident occurred thirty-six hours before admission. He gave a history of scaphoid fracture fifteen years before. On admission he showed a crack through the radial styloid without displacement and the old fracture of the scaphoid with nonunion and osteoporosis. He had had only slight pain with the old scaphoid lesion over the fifteen-year period, particularly in bad weather or in particularly heavy work, but was able to do the heavy work of an automobile mechanic. He was treated by strapping support and gradually increasing active exercise. At the end of three weeks he had full motion range with only slight aching and tenderness. At five weeks he had full function, occasional ache, said his wrist felt somewhat weak but was back at work as an automobile mechanic.

At three months it was noted that there were slight pain and stiffness on waking in the morning and in bad weather, but not sufficient to interfere with his regular work. He had normal motion range except for 10° loss in palmar flexion and some definite tenderness in the snuff-box.

In November, 1930, sixteen months after the above injury, he struck a man on the head during a fight and came to the Fracture Clinic again, with classical signs of a fracture of the scaphoid, but no evidence of fresh fracture by X-ray. After a month of treatment, with persistent symptoms, he entered the Presbyterian Hospital, where, on December 17, 1930, Doctor Murray removed the whole scaphoid through a dorsal incision under avertin anæsthesia. He healed by primary union, active function was started on the fifth day without subsequent immobilization, and during these five days physiotherapy was used on the wrist and hand. Fourteen days after operation he was back at work, being bothered only when he tried the heavier work, which he could not manage. He then had full pronation and supination and could extend to 200° without pain and flex normally. A wrist strap was advised.

On January 19, 1931, it was noted at follow-up that he was on full work but had to safeguard the hand in the heavier labor. His only disability was in strength of grip and in dorsiflexion, which is still limited, but the man is doing full work. It is now a little over two months after operation. His only difficulty is with extremely heavy lifting for which he does not feel he has sufficient strength in the wrist.

RETROPERITONEAL FIBROMA

Dr. WM. BARCLAY PARSONS, Jr., presented a woman, fifty-four years of age, who was admitted to the Presbyterian Hospital February 21, 1929, complaining of severe cramp-like abdominal pain, which had persisted for two years, associated with great loss of weight and strength. Her previous health had been good. There had been two miscarriages, and an operation for appendicitis eight years prior to admission. About five years before admission she had a fall and was supposed to have strained her hip. Following this incident she began to fail—that is to say, she had malaise, lost weight, and was unable to do her work. During the next three years she dropped from 160 to 106 pounds. This loss of weight seemed to be consequent on her loss of appetite, and no other definite symptoms during this period were elicited. Two years before admission, severe cramp-like abdominal pain appeared, starting in the umbilical region and radiating to the flanks with particular accentuation in the right lower quadrant. Frequently the pain would appear about two hours after meals, but this relationship was by no means constant. There was marked associated constipation with definite abdominal distention. Rich, fatty, and bulky foods had a tendency to precipitate the attacks, which were at times relieved by eructation of gas. Relief on taking food was never apparent. During the few months just before admission she would average five attacks a day, each one lasting five or ten minutes, of a severe and exhausting degree. There was marked anorexia, but no nausea, vomiting, nor jaundice. Stools had been hard and small, and she had used castor oil and enemata frequently with some relief. During the two years prior to admission there had been a further loss of weight from 106 down to 83 pounds, representing a total weight loss of nearly 80 pounds.

Examination on admission revealed a poorly nourished individual moaning and rocking with pain. Her face was flushed and damp with perspiration. Aside from the evidences of emaciation, a soft blowing apical systolic murmur, and a blood-pressure of 178/96, the only physical findings of note were in the abdomen, which was markedly distended; in the right lower quadrant

a firm mass was readily palpated. It lay apparently mesial to the cæcum, was hard, nodular, and fixed.

She was observed for nearly three weeks before operation, during which time there was marked abdominal distention requiring frequent enemata and colon irrigation. Associated with the distention there was considerable pain, sometimes radiating to the shoulder, sometimes down the leg.

At operation, which was done March 12, 1929, under spinal anæsthesia, there was found a lobulated, firm, yellowish-white tumor measuring 12 by 8 by 6 centimetres in the retroperitoneal tissue behind the mesentery of the terminal ileum and ascending colon. The mass was well encapsulated. The right ureter lay deep in the fissure between two of the bosses of the tumor, and was definitely narrowed for a distance of perhaps 2 centimetres; but it was readily dissected free from the tumor and its wall was not compromised. The cæcum and ascending colon were liberated by a lateral incision and displaced mesially. The tumor was excised, the peritoneum was repaired, and the abdomen was closed in layers.

Microscopic examination of the specimen established a diagnosis fibroma of the retroperitoneal tissue.

The post-operative course was marked by severe chronic gastric dilatation requiring lavage and the use of a duodenal bucket for over three weeks. An X-ray taken four weeks following operation showed a markedly dilated atonic stomach, as compared with a medium position with normal tone and regular peristalsis in the examination prior to operation.

Since discharge a gain in weight has been steady and satisfactory. At the end of one year she had gained 50 pounds.

Doctor Parsons remarked that most benign retroperitoneal tumors are lipomatous, and that when fibromas occur they are apt to be found within the leaves of the mesentery. This is the only case of retroperitoneal fibroma in the surgical pathologic laboratory at the Presbyterian Hospital. The typical mesenteric tumor presents mobility as one of the striking physical findings. Due to its situation and close relationship to the ureter, this mass was immobile, and for that reason suggested the more frequently encountered sarcoma. It is somewhat difficult to explain her severe pain, distention and loss of weight, unless there was both a vagus and sympathetic interference. The associated extreme loss of weight was also suggestive of malignancy.

Dr. Frank S. Mathews said he once had shown before this Society a patient—a man—from whom he had removed a retroperitoneal fibroma weighing eleven pounds. The patient had been unconscious of its presence and symptomless until shortly before admission to the hospital, when he developed severe abdominal pain. The tumor was easily lifted out with a coil of small bowel over it which required resection with the tumor. The patient was seen five or six years after operation, which confirmed the diagnosis of fibroma as contrasted to fibrosarcoma.

TUBERCULOSIS OF PERITONEUM, FIFTEEN-YEAR RESULT

Doctor Parsons presented a woman, aged twenty years, who was admitted to the Presbyterian Hospital April, 1916, with a two years' history of general cramp-like abdominal pain gradually increasing in frequency and severity until there had been constant pain for two weeks prior to admission. During these weeks there had been a noticeable increase in the size of the abdomen.

with some fever at night. Her appetite was poor. There were no disturbances in defecation, urination, nor menstruation. She had been married for two years. There had been no pregnancies. In her previous health there had been no serious illnesses, no accidents and no operations, and there

was no history of exposure to tuberculosis.

She presented, when admitted, a chronically ill appearance, with no evidence of lung involvement except loss of resonance because of abdominal pressure. The abdomen was markedly enlarged, with every evidence of the presence of fluid, including a readily elicited wave; but there was no change in the fluid level following change of position of the patient. The abdomen was so tightly distended that no masses or viscera could be palpated, but the cervix was definitely pushed down and was moderately tender. She was observed for four weeks prior to operation, during which time she ran a temperature ranging between 100° and 103°. Abdominal paracentesis was performed twice during this period. A laparotomy, done May 3, 1916, revealed the peritoneum much thickened, and studded everywhere with innumerable miliary tubercles. The parietal peritoneum was particularly thick; omentum matted over intestines. Beneath omentum was a large collection of almost gelatinous yellow fluid and a large quantity of a mushy, grayish The intestines were heavily matted together and could not be separated without much bleeding. Pelvis as well as upper abdomen was similarly filled with matted coils of intestines, so that it was impossible to determine whether tubes and ovaries were involved in the process or not. With the finger an opening was made through the omentum and its adhesions, and the fluid contained within was allowed to escape. Hot saline solution was poured in and sucked out. Pieces of this omentum and adhesions was taken for a specimen, as was a piece of the parietal peritoneum.

Microscopically the section from the peritoneum showed a mass of connective tissue containing innumerable tubercles, a number having several giant cells and large areas of caseation. Section from tissue, thought to be a gland, showed no glandular tissue, but cedematous fibroblastic tissue containing tubercles along one edge. Section of gelatinous material from peritoneal cavity shows mainly amorphous material containing some cell detritus

and leucocytes.

The patient made a slow convalescence. She was discharged to the country two months after operation. Three or four months later she was drained again in the Convalescent Hospital, and the wound healed four or five months later. She returned to the Follow-up Clinic complaining of a median ventral hernia. In May, 1923, seven years after the first operation

she was admitted for repair of the ventral hernia.

When the abdomen was opened, the following conditions were found: A wide diastasis of the recti and a deficiency in the anterior sheath. There were numerous vessels and bands covering the small and large bowels. In the pelvis, there were numerous adhesions binding small gut to uterus, tubes and ovary. In some of the adhesions and in the peritoneum covering the large and small bowels in many places in the mesentery and in a few places in the parietal peritoneum were small, rounded, yellowish bodies varying from I to 3 centimetres in diameter. These were undoubtedly tubercles, but whether fresh or old could not be told. There was a small amount of free yellowish fluid in the peritoneal cavity. The condition of the pelvic organs could not be determined on account of adhesions but the uterus was apparently plastered up in the anterior position. There was an enlarged mass on the right side thought to be right tube and ovary.

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After the removal of specimens a horizontal overlap was done. Microscopic examination of the specimens was reported as follows:

"Sections of the small bit of tissue with a cyst at one end of it show that the cyst is very thin-walled and is lined with a single layer of very small flattened endothelioid cells and is filled with red-staining amorphous material. Attached to one end of this is some loose tissue containing masses of hyalinized scar tissue resembling the corpora albicantia of the ovary. The surrounding connective tissue is very loose, contains many small dilated capillaries and some blood pigments. At the end opposite the cyst is an area of epithelioid cells with some capillaries mixed in. The other small rounded bit of tissue is fibrous and contains many large and small rounded spaces which are surrounded by apparent syncytial masses. There are a few giant cells with several small vacuoles in the cytoplasm. In some areas there are numerous capillaries.

The peritoneal fluid was injected into a guinea pig on May 14, and the pig was killed on June 25. No evidences of tuberculosis were found in the lung, liver and spleen.

The patient made an uneventful recovery. For the last six years she has been happy and contented. She has gained weight, having been over 190 pounds for the last four years.

EXOPHTHALMIC GOITRE: PERSISTING UNILATERAL EXOPHTHALMOS: SECTION OF CERVICAL SYMPATHETIC

Doctor Parsons presented an adult man who was admitted to the Presbyterian Hospital in March, 1927, for a partial thyroidectomy for exophthalmic goitre. The main complaint had been exophthalmos and poor vision for eight or ten years. The other symptoms of hyperthyroidism were moderate and had been present three or four years. The striking feature in his physical examination was the exophthalmos.

Following the first operation there was a critical post-operative period due to Type IV bronchopneumonia, from which he made a prompt and satisfactory recovery. There was a noticeable improvement in the appearance of the eyes following operation, but particularly in the left eye. He returned to active work handling freight three months after the operation, and has continued at it since, the metabolic reading being normal at various times.

He was readmitted to the hospital in March, 1929, because of unilateral exophthalmos. On the suggestion of Dr. Byron Stookey, a section of the right cervical sympathetic was planned to induce the syndrome described by Horner in 1876, consisting of miosis, enophthalmos, pseudoptosis, and interruption of sweating. This syndrome is a well-recognized one, and follows lesions of the cervical sympathetic.

Prior to the operation, several observers on different occasions noted a 3-millimetre difference in the position of the two eyeballs, the right exhibiting a greater degree of exophthalmos. They also agreed that the width of the right palpebral fissure was 3 millimetres wider than the left. Operation was done March 15, 1929, the right cervical sympathetic being divided at the upper margin of the superior ganglion.

On discharge there was the same difference in position of the eyeballs noted by exophthalmometry. The right side of the face was dry. The right pupil was contracted and did not dilate with cocaine. There was an immediate ptosis produced, giving a striking improvement in appearance; and on subsequent examinations up to June, 1930, the right fissure has been equal to, or I millimetre less than, the left fissure. The last exophthalmometry showed that the discrepancy in position between the two eyeballs had diminished slightly, but the right was still 1½ to 2 millimetres more prominent than the left.

The same failure to produce enophthalmus was noted in another identical case, and in two others presenting Horner's syndrome except the enophthalmos. One of these was a patient from whom Dr. Frank Meleney removed the cervical sympathetic ganglia for chronic arthritis, the other being a patient with metastases to the cervical lymph-glands following carcinoma of the breast. There is no third nerve paralysis. The ptosis due to paralysis of the tarsal muscle is really a pseudoptosis. The enophthalmos included as part of this syndrome is more apparent than real. It would be of interest if further data on this point were collected. The mechanics of exophthalmos are as yet unknown, but it would seem that the sympathetic nervous system probably represents the pathway by which it is accomplished. It is such a striking feature and so disfiguring, and when extreme represents such a severe hazard, that any effort to correct it is well worth undertaking.

In this case, striking and satisfactory improvement in appearance was obtained by the production of ptosis. This operation is indicated in cases of unilateral widening of the fissure. The speaker would hesitate to use it on both sides, because the miosis might conceivably interfere with night vision. It is not to be considered as a substitute for plastic procedure on the lids in those cases where the exophthalmos is so severe that the lids fail to close at night, and corneal ulceration has begun. This circumstance represents a real emergency and must be handled as such, whereas the procedure suggested in this report is merely for cosmetic purposes when there is a marked difference in the appearance of the two eyes.

Dr. Emil Goetsch said that he had done this operation in several of these cases and the result, as Doctor Parsons reported, was always effective. The relief of the exophthalmos is nearly always the same. Some of these have to be done because of the extreme exophthalmos and the danger of corneal ulcer that every now and then occurs. The same effect of enophthalmos and small pupil is seen following this procedure in angina pectoris, but it is a very nice procedure for relief in these extremely severe cases. In most of his cases, Doctor Goetsch had considered the opposite eye as somewhat more prominent than it should be. He had seen only one or two cases of true unilateral exophthalmos in which there was no protrusion or enlargement at all of the opposite eye. In most of the so-called instances of unilateral exophthalmos one eye is simply more prominent, though one cannot say that there was no prominence whatever on the opposite side.

Doctor Parsons, in closing the discussion, said that in the cases where he had done sympathectomy, enophthalmos did not follow, and stressed the fact that improvement in appearance was due to paralysis of the tarsal muscle.

AN ANALYSIS OF THE MORTALITY IN A SERIES OF SEVENTEEN HUNDRED AND FIFTY-FIVE OPERATIONS FOR GOITRE DURING A TEN-YEAR PERIOD, 1920 TO 1929, INCLUSIVE

Dr. Emil Goetsch read a paper with the above title, for which see page 167.

Dr. Chas. Gordon Heyd was impressed with the fact that the mortality rate following thyroidectomy for hyperthyroidism is essentially the same as

that for chronic appendicitis and approximately 40 per cent. better than what obtains by and large throughout the country in operations for acute appendicitis. He had always felt that the basal metabolic determinations were not trustworthy indications as to operability. A young girl of twenty with a basal metabolic rate of 60 was a far better risk than a patient of sixty with an auricular fibrillation and a basal metabolic rate of 18. In regard to ligation, he had not performed a single ligation in the past year and believed its indications were rapidly passing away. He had felt that the ligation carried with it intrinsic dangers and in addition to that it accounted for some patients leaving his service. A ligation should never be done without the definite promise upon the part of the patient for a resection eight to twelve weeks later. Some patients feel so much improved after ligation that they do not return. It has been the habit in his clinic to do a bilateral resection. A single resection would constitute about 2 per cent. of their work and they had been largely influenced as between a single or double resection by pulse response during the course of operation. It seemed to Doctor Heyd that the important element in Doctor Goetsch's paper was the broad principle that in goitre operations there was represented to the laity an operation of tremendous risk and yet throughout all the clinics in the country, with a standardized preparation, a standardized technic, it was one of the least dangerous of the major operations. Doctor Heyd believed the largest factor in this lessened mortality was the adequate pre-operative therapy. It was unfortunate that most patients who come to the clinic had been iodinized and many had received what were tremendous doses of iodine for a long period of time. In the pre-operative treatment he emphasized (1) the necessity of complete and absolute rest in bed; (2) transfusion, both as a food and as blood replacement; (3) overcoming dehydration by daily intake of at least three to four thousand cubic centimetres of water, the giving of a highly concentrated, caloric diet, with adequate amounts of glucose by rectum, and finally, where the patient had not been receiving iodine, a short intensive course of Lugol's solution. In regard to anæsthesia, he had discontinued local anæsthesia as the psychic trauma was a greater handicap to the patient than were the benefits from the anæsthesia per se. In their clinic they had used amytal intravenously, or large doses of luminal, to produce twilight sleep, supplemented by ethylene or nitrous-oxide gas anæsthesia, and more recently occasionally have used avertin by rectum.

Dr. William B. Parsons, Jr., said that he thought that the patients' mortality of 2.16 per cent. would have been diminished if they had had the benefit of iodine in the early cases where ligation was done. He believed that there is an important place for the two-stage procedure; there are a certain number of patients who cannot be prepared with a satisfactory degree of safety for a one-stage thyroidectomy. Ligation in itself is of no value as far as the individual is concerned. The good that occurs is due to the prolonged rest, proper dietary régime and other accessory features. The stage

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procedure should be decided upon before the patient reaches the operating room and one should not wait for signs of danger to occur before changing from a bilateral to a unilateral lobectomy.

Dr. Morris K. Smith asked Doctor Goetsch whether in his experience the patients whom he described as presenting a somewhat deceptive appearance of well-being after indiscriminate thyroid medication showed danger signals on the operating table, or whether their condition was first manifested by alarming post-operative reaction.

DOCTOR GOETSCH, in closing the discussion, said that he was of the opinion that there were cases where one dared not do more than ligation. He believed one use of the ligation operation was this: The patient has been going from physician to physician with nothing but the indiscriminate use of iodine therapy and he has become disgusted. If he is asked to rest and go through a period of preparation for operation, he refuses; he wants something done at once. If there is nothing more in doing ligation there is the satisfaction of getting control of the patients, for after this they do what they are told. Doctor Goetsch did not believe that the benefit following ligation came only from rest, dietary régime, etc. He had many patients who had gained from ten to thirty pounds and some were so well that they did not want to come back for further procedure, but he never let the patient go with ligation alone if he could help it. One should tell them at the time that they will have to go through the resection operation for if one waits too long the operative dangers are much greater. They should not go longer than six weeks to two months. Answering Doctor Smith as to the iodinized patients, there was no warning at operation or soon after and that is the reason Doctor Goetsch had carried on and not worried. One patient had been receiving iodine for a year but was in such good condition that a good prognosis was made and a double resection was done, but the patient died in six hours without giving previous warning of danger.

STATED MEETING HELD MARCH 11, 1931 The President, Dr. Edwin Beer, in the Chair

EXOPHTHALMIC GOITRE—RESULTS FOLLOWING RADIATION THERAPY AND SURGERY

Dr. Arthur S. McQuillan presented a woman, thirty-six years old, whom he first saw in May, 1928, when she gave a history of having had exophthalmic goitre for a year previous. At this time, there was evidence of loss of weight, bilateral exophthalmos, fine digital tremor, flushed skin, average of three bowel movements a day, pulse 136, feeble, with occasional skips. The thyroid gland showed only a slight symmetrical enlargement. It was quiet and soft. She had had rest and iodine feeding during most of this year. Surgery was advised, but patient refused. Eighteen months later the woman was again seen. She had remained in bed six weeks after first visit, and at intervals thereafter up to a month previous had eighteen sets of X-ray treatments (a set meaning an exposure on each lobe and on back of neck). She had increased in weight, from 117 to 130 pounds; had good

endurance, good appetite, and slept well. However, she had attempted to return to her work as a stenographer, but had to discontinue owing to rapid heart, tremor and nervousness. She improved with rest and tried working again with same result. Metabolism determinations during this time were never below 25 per cent. plus. Physical examination at this time showed weight 132 pounds, blood pressure 155/80, pulse 120, heart not forceful but premature systole every four beats, moderate bilateral exophthalmos and no visible or palpable thyroid gland, and basal metabolism of 23 plus.

The thyroid was removed. A gland less than normal size was found. Capsule was adherent, tissue was pale in color with shriveled veins on surface. The tissue was avascular, fibrous and on cutting had consistency of cartilage. (Fig. 4.) About one-third of each lobe with isthmus was resected. The recovery was quick. One month after operation pulse was 80 and regular. There was a gain of five pounds and metabolism normal. Two months later, patient resumed work and has lost no time since. At present, the patient is normal with exception of perceptible exophthalmos.

Dr. Alexis V. Moschcowitz stated that his experience differs somewhat with that of Doctor McQuillan's. He had operated on cases of Graves' disease which had received radiation, but found that in spite of the radiation, the bleeding was just as extensive. Nor had he noticed any special benefit from radiation, as the basal metabolism was reduced very little, if any, by this procedure. Doctor Moschcowitz also called attention to the fact that in order to obtain material benefit from resection, he is much more liberal in the amount of resected tissue than is Doctor McQuillan.

Doctor McQuillan rejoined that he had never seen permanent and complete relief resulting from radiation in exophthalmic goitre. But, however, there was no doubt that temporary results were obtained. There were more and more cases undergoing radiation therapy with the idea of a permanent cure. This is to be condemned, because cure is not accomplished and makes subsequent operative procedure much more difficult. Doctor Moschcowitz stated that he could not get permanent relief by resecting so small an amount of thyroid tissue. Doctor McQuillan stated that the resection in this case in no way represented the amount of resection in the usual case, as in this case the gland was less than normal size, due to its great damage with scar tissue. Doctor McQuillan felt there might be damage in resecting more than one-third of each lobe with isthmus in this particular case.

Doctor McQuillan presented also a girl who was admitted to Bellevue Hospital, December, 1929. She was eleven years of age, and gave a history of a goitre since the age of seven. The goitre had always been present on both sides of neck and had gradually increased in size to time of admission to the hospital. It had given the patient no trouble until two months before admission, since which time there had been increasing dyspnæa, intensified at times by change in position of head. There had been no loss of weight or other signs of hyperthyroidism.

On admission, there was definite and marked dyspnæa, hoarseness and occasional cough. Breathing, especially inspiration, was stridulous and audible some distance from patient. She was pale and at times slightly cyanotic, slightly stooped and bore an anxious expression. Generally there

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was underdevelopment both mentally and physically. X-ray gave further evidence of tracheal compression from lateral aspects.

The goitre, ten times the size of a normal thyroid gland, was made up of multiple nodules, none showing cystic change. A resection was done, leaving an amount of thyroid tissue on either side equal to that of a normal thyroid gland. The trachea was found to be compressed laterally, and the compression deformity persisted to a marked degree after the tissue mass causing the pressure had been removed. Before the operative procedure and during the primary anæsthesia a No. 17 silk catheter was introduced into the trachea beyond the point of obstruction, to safeguard against complete block of air passage during the surgical manipulations of the goitre. The operative procedure was without difficulty. Two noteworthy observations were the small size of the trachea in this underdeveloped girl and the high level of the dome of the pleura in the neck, which is usual in children.

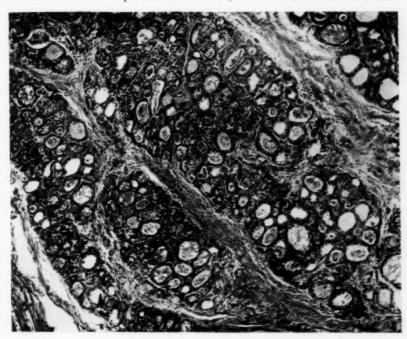


Fig. 4.—Section of thyroid tissue from a case of exophthalmic goitre, showing the result on thyroid tissue of exposure to X-ray over a prolonged period. (Eighteen exposures over a period of several months both sides and back of neck.) Gland tissue is seen divided into small islands of hyperplastic tissue, these islands separated by dense fibrous tissue and round-cell infiltration.

Convalescence was smooth with the exception of the second twenty-four hours. During this time tracheal obstruction recurred to an alarming degree, accompanied by stridor and cyanosis. Changing the position of the head, however, relieved the obstructive breathing, the neck being put in hyperextension. This exerted pull enough to relieve the obstruction in the partially collapsed trachea, the mucous membrane of which was undoubtedly congested and filled with mucus. With the additional treatment of morphine-atrophin-benzoin-steam inhalations, the condition rapidly cleared and the patient has enjoyed good health to the present time.

Dr. Henry W. Cave was reminded of an unusual experience he had had in France during the War. A young man of twenty-two in the influenza

ward one morning on awakening stretched his arms up, pulled his head back and coughed; thereafter he could not get his head down and was rapidly turning blue. The nurse called for help; and Doctor Cave, examining the man, found he had an adenoma of the isthmus of the thyroid, a substernal affair, and that this movement of stretching his arms up and putting his head back had dislocated this adenoma forward and upward. He was rushed to the operating room and on incision a cystic adenoma of the isthmus was found and enucleated. The patient promptly recovered.

SUPRACONDYLAR FRACTURE OF HUMERUS

Doctor McQuillan presented a woman, fifty-seven years old, who, October 23, 1930, fell, striking directly on her elbow, sustaining a supracondylar fracture of her right humerus, the condyles slightly wedged apart and a (T) line of fracture extending into joint, with practically no deviation in the lateral or anterior-posterior axis. The arm was put up in moderate flexion, with forearm in supination. X-ray in this position showed the fragments in fair position, so that further manipulation was considered unnecessary and hazardous in view of the nerve relations about this joint.

Manipulation revealed union of fragments at end of three weeks, and

X-ray showed callus at the end of six weeks.

The final result has given pronation and flexion practically normal. Supination limited 25 per cent., and extension limited 10 to 15 per cent. The patient states she has practically full recovery of former function, the present impairment was present before injury, and exists to an equal degree in the other elbow-joint, due to a deforming arthritis, which she has had for several years.

RECURRENT RUPTURED GASTRIC ULCER—GASTROENTEROSTOMY SYMPTOM-FREE FOUR AND ONE-HALF YEARS

Doctor McQuillan presented a man, forty years old, who, May 29, 1923, was admitted to Bellevue Hospital with diagnosis of perforated duodenal ulcer. The perforation had apparently taken place ten hours previous to admission. Operation confirmed diagnosis with exception that the perforating ulcer was on anterior pylorus near the lesser curvature. There was fluid throughout the upper peritoneal cavity. The patient's general condition was none too good. The perforation was closed over and the wound closed with drainage. There was nothing noteworthy about convalescence. The patient gave a typical ulcer history over a period of three years previous to this hospital admission. There was much epigastric pain and distress for two weeks previous to perforation and a history of vomiting and passing of blood by rectum, one week before.

Following this operative procedure patient was under observation for about eight months and during this time there was steady improvement and no ulcer symptoms. However, gastroenterostomy was advised. Patient was not heard from until December 15, 1925, when he was admitted to Bellevue again with a similar diagnosis. From the history the perforation was about four hours old, but the patient was in poor condition, having lost much weight and having suffered from ulcer symptoms for three months previous. At operation the perforation was found in the region of the old scar. There was a pouch of fluid walled off by omentum. The patient took the anæsthetic poorly and due to his general condition, no procedure other than closure

seemed justifiable. Recovery again fortunately was slow but not complicated. For the next six months the patient was treated by rest and diet, with the result of twenty-five to thirty pounds' gain in weight, and ulcer symptom-free. In June, 1926, seven months following the second perforation, a posterior short loop gastroenterostomy was done. At this time there was only slight scarring at the site of the previous perforations and no definite crater like ulcer could be identified. Patient made a good recovery and has maintained his weight and has been symptom-free to present time. (Four and one-half years.)

DIVERTICULUM OF DUODENUM

Dr. William H. Barber presented a woman, aged twenty-four years. who was admitted to Bellevue Hospital October 13, 1930. She had undergone an appendectomy in 1919; had been operated upon for gastric ulcer in 1925 at which time a pyloroplasty was done. She suffered from an internal hæmorrhage in 1929 for which she entered a New York hospital and received medical treatment as for duodenal ulcer. Since 1917 she has suffered from pain in lower epigastrium which pain is relieved by soda bicarbonate and vomiting; it is not aggravated by eating. Since her 1925 operation she has had dull, aching pain in her back. X-ray examinations after admission indicate a large duodenal diverticulum with pylorospasm together with adhesions about the pylorus. The diverticulum sprang from the first part of the duodenum, inferior border; Wassermann negative. November 12, 1930, the abdomen was opened; stomach negative excepting for adhesions about the pylorus which were massive. No attempt was made to identify the diverticulum. A posterior gastrojejunostomy was done. The post-operative treatment included two transfusions. At the present time she is free from pain and has increased in weight.

SYPHILIS OF BREAST

Doctor Barber presented a woman, aged thirty-eight, who was admitted to Bellevue Hospital, November 10, 1930, on account of pain in her right breast, of two months' duration, accompanying an ulcer just below right nipple. (Fig. 5.) Her family and previous history were irrelevant. She stated that two months before she had received a blow in right axilla without suffering any discomfort at time. One month later she observed a scab at outer right margin of nipple, not at site of trauma previously reported. At same time she began to suffer intermittent pain in breast and in right axilla. She received miscellaneous treatments from physicians without relief. Upon removal of scab a slight purulent discharge was observed and an ulcer remained.

When admitted there was an irregularly demarcated ulcer involving areola and outer edge of nipple with an area of infiltration extending slightly beyond the limits of the ulceration and measuring approximately $2\frac{1}{2}$ by $3\frac{1}{2}$ centimetres. Floor of ulcer made up of granulation tissue which bled freely. Glands in right axilla enlarged, discrete, intact but not as hard as those typical of metastasis. Right breast otherwise negative. Left breast—slightly thickened.

Laboratory Data.—Wassermann (blood)—4 plus (on 16th and 26th Nov.) Blood.—White blood-cells 7700; Polymorphonuclears, 2 per cent.; lymphocytes, 18 per cent.; red blood-cells, 410,000; hæmoglobin, 85 per cent. Urine.—Negative for albumen, glucose, casts.

Progress.—She was referred to the service of Dr. M. B. Parounagian, Bellevue Hospital, November 28, 1930, who reports as follows: "In view

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of the short duration, clinical appearance and Wassermann 4 plus, also the ready response to antisyphilitic medication I am led to regard the condition as a primary lesion of lues. Treatment from November 28 to date consisted of eight neo-salvarsan and twelve mercuric salicylate injections. The results are very satisfactory. She will have a rest period and resume treatment again."

Dr. Charles L. Gibson said that he had never seen but one authentic case of syphilis of the breast. The patient was a young Italian woman, married, who came in with an indefinite history. There was massive infiltration of the breast which was irregular in shape and hard. It did not look like anything that Doctor Gibson had ever seen before. The patient was closely questioned and the fact was brought out that although she had been pregnant several times she had never had any living children. Wassermann examina-



Fig. 5.-Syphilis of breast.

tion disclosed the true condition and antiluetic treatment cleared it up completely.

Dr. Frederick W. Bancroft said that he recently saw a case of syphilis of the breast in a young woman nineteen years of age of good family. She came in with the diagnosis already made. There was absolutely no venereal history and no history of trauma. There had never been a secondary lesion. She had a small ulceration of the nipple which would not heal. The Wasserman reaction was 4 plus and the Kahn 4 plus, and under antiluetic treatment the condition rapidly cleared up.

Dr. Frank E. Adair said that he had treated two cases of syphilis of the breast. As a rule the *primary* chancre of the nipple does not come to the surgeon; nor do the *secondary* lesions—the mucous patch and the skin eruption. It is the forms of the *tertiary* lesion that fall chiefly into the practice of surgeons and are commonly confused with carcinoma of the breast.

SARCOMA OF KIDNEY

In general, there are two forms of the tertiary lesion—the gumma and the subcutaneous form. In 1924,¹ going back over the literature for 155 years, Doctor Adair was able to collect forty-five authentic cases of gumma of the breast and to this report added one case of his own. Again, in 1929, a case of the "subcutaneous type" which simulated Paget's disease was reported by Pack and Adair.² Virchow, in 1861, said: "Concerning syphilitic tumors of the breast we know very little." He furthermore stated that he would welcome an opportunity to study one. In the 3881 cases with breast lesions at the Memorial Hospital during the twelve-year period, January 1, 1927, to January 1, 1929² there were but two tertiary syphilitic lesions, giving an incidence of 0.05 per cent. It is therefore a very rare lesion and one which is probably often overlooked on account of its rarity. Any clinician who makes the diagnosis is to be congratulated.

REFERENCES

Adair, Frank E.: Gumma of the Breast: Its Differential Diagnosis from Carcinoma. Annals of Surgery, pp. 44-54, January, 1924.

² Pack, George T., and Adair, Frank E.: Tertiary Syphilis of the Breast. Arch. of Derm. and Syph., vol. xx, pp. 806–810, December, 1929.

⁸ Pack, George T., and Le Fevre, Robert G.: The Age and Sex Distribution and Incidence of Neoplastic Diseases at Memorial Hospital, New York City. Jour. Cancer Research, vol. xiv, pp. 167–294, 1930.

SARCOMA OF KIDNEY

Doctor Barber presented a girl, aged twelve, who was admitted to Bellevue Hospital August 23, 1930, for treatment of a mass in abdomen which had been noticeable for three years. (Figs. 3 and 4.) It was hard and located in right hypochondrium at subcostal border. One week before admission complained of pain, Gastro-intestinal and urinary symptoms negative. Temperature ranged from normal to 102°. This mass was visible in the right upper quadrant and extended over into the left half of upper abdomen and also into right costovertebral region. It was nonpulsile, descended on respiration. Occupied the entire right upper quadrant and was traceable dorsad into region of right kidney. (Figs. 6 and 7.) Spleen not palpable. No evidence of fluid in abdomen.

Wassermann—negative. X-ray—December 12, 1930. Chest: Isolated calcified focus axillary portion right first interspace. No active infiltration.

Operation.—August 23, 1930, under general anæsthesia. Findings.— Large mass covered with vascular peritoneum and intimately connected with the undersurface of right lobe of liver, posterior abdominal wall and involving the greater portion of right kidney. It extended downward to the level of the iliac crest and mesial to the outer border of the left rectus. It was lobulated and appeared to fluctuate. The free portion of the tumor dislocated left lobe of liver and gall-bladder into left abdomen. There was no free fluid in abdomen. Liver appeared normal excepting for change in position. Pancreas and left kidney were normal. Stomach and duodenum not involved.

Technic.—Right rectus incision 10 centimetres in length. After exploration the probable diagnosis appeared to those present to be echninococcus cyst springing either from liver or kidney. To prevent contamination of peritoneum, edges of wound were closed about an exposed portion of tumor, as a preparation for marsupialization.

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September 3, 1930.—Exposed portion of tumor was biopsied. No fluid but solid tumor mass was found. Repeated aspiration into mass disclosed nothing but blood. Wound was dressed. Patient returned from operating room in good condition.

Microscopic Report by Doctor Symmers was as follows: "Microscopic examination (Figs. 8 and 9) shows the presence of a malignant tumor composed of blood-vessels lined by endothelium and surrounded by a thick mantling of lightly staining cells of the fibroblastic type, the latter showing sudden transition into richly chromatic spindle-shaped tumor cells arranged radiately to the long axis of the vessel lumen and growing profusely, sometimes continuing the radiate arrangement, at other times arranged circumferentially or growing diffusely. The tumor belongs in the general category with the so-called peritheliomata or angiosarcomata. These tumors are, as a rule, extremely malignant, grow rapidly and metastasize widely. The prevailing opinion seems to be that they are derived directly from the connective tissue of the blood-vessel wall."





Fig. 6.—Abdominal mass. Transverse aspect.

Fig. 7 .- Abdominal mass. Anterior view.

Post-operative Care.—In addition to supportive treatment including transfusions she has been receiving X-radiation therapy from Dr. Ira Kaplan from September 4, 1930, to the present time. By March 20 she will have received two series of treatments and Doctor Kaplan commented as follows: "Tumor mass did not seem to have regressed at all. One feels that this tumor is not radiosensitive. Treatment has been started again."

General Condition.—Patient on repeated hospital visits seemed at first to have improved but later to have become progressively worse with increase in size of mass and a slight loss of weight. She has been attending school irregularly up to past week.

LATE RESULTS OF SIMPLE SUTURE IN ACUTE PERFORATED DUODENAL ULCER

Dr. WILLIAM CRAWFORD WHITE read a paper with the above title, for which see page 242.

SIMPLE SUTURE IN ACUTE PERFORATED DUODENAL ULCER

DR. CHARLES L. GIBSON said that for many years surgeons have held opposing views of what should be done in the presence of acute perforated

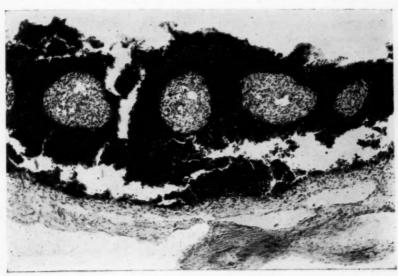


Fig. 8.—Sarcoma of kidney. Low-power section.

ulcer. Doctor White's paper is based on the important factor of end-results, and he had raised one or two points on which Doctor Gibson wished to com-

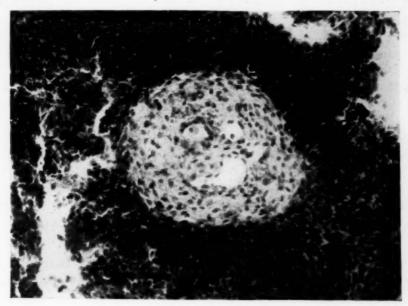


Fig. 9.—Sarcoma of kidney. High-power section,

ment. Doctor White had been skeptical of Doctor Gibson's theory of referred pain. As a matter of fact, it is not readily determined. The history of it must be searched for. This referred pain, somewhere in the chest above the clavicle, comes on not a long while after the original pain but about a half-hour after the great pain in the abdomen. It is quite transitory and disappears in one-half to three-quarters of an hour. Meanwhile, the pain in the abdomen still persists and so completely occupies the patient's attention that he does not notice the lesser pain. When convalescing they sometimes remember that they had it but that it went away. Most hospital histories, being deficient, seldom record this fact. In border-line cases, it is a great help to know of this added symptom. It can be elicited in between one-third to one-half the cases. Another point Doctor White had not referred to was the obliteration of liver dullness. If there is considerable gas in the abdomen, this will be seen under the fluoroscope but in many there is not a bubble of gas except in terminal cases, and those do not belong in this discussion. In regard to the fact that many of these cases heal so well and have so little trouble later, the perforation having apparently cured them, this seems logical. When the slough separates, they will heal. They do heal and are cured by the process of perforation. Sometimes in closure, one must produce some form of constriction and Doctor Gibson has adopted the plan of not using purse-string sutures but using interrupted sutures on the Heineke-Miculicz principle. These cases have healed up very well. The speaker has never done anything but closure of the ulcer in acute perforation except on one case and that could not be closed and resection had to be done. He has had many of these cases and has tried to keep them under observation. The mortality has been between 16 and 18 per cent. and has been so constant that it seems as though it has been stabilized. Fifteen per cent. have had to have some secondary operation such as gastroenterostomy or other procedure. As to whether the operation should be supplemented by something else: A good surgeon may do a gastroenterostomy but that this should be a dogmatic formula is objectionable. Gastroenterostomy, if it is done at all, should be done well, and this is not possible with every surgeon. Doctor Gibson subscribed to every word Doctor White had said in this respect and believed that in the average case one should resist the temptation and let the patient get well with a simple operation. With regard to the wave of radical surgery being done in continental Europe: Doctor Gibson quoted Hartmann's figures collected in Paris: Simple suture, 33 cases, deaths, 22 per cent.; excision with suture, 19 cases, 5 deaths, 21 per cent.; gastroenterostomy, 20 cases, 8 deaths, 35 per cent.; resection, 19 cases, 5 deaths, 20 per cent. These figures show 33 simple to 58 more complicated procedures, which indicates what they are doing abroad.

Dr. John E. Jennings agreed practically entirely with Doctor White's point of view. He had not found it necessary to do gastroenterostomy as a routine; even in cases with large perforations and threatened obstruction he would rather not do it if he could avoid it. The reference made by Doctor Gibson to the occurrence of a clavicle pain was interesting. The speaker had noticed it in a number of cases. It had appeared to him to be analogous to the pain one gets in a ruptured ectopic pregnancy and associated with fluid in

the lesser sac. This should be carefully aspirated with a suction tube or similar apparatus. High elevation of the head of the bed has had some value, particularly in early cases and in late cases with peritonitis. Even early cases, before six hours, require sufficient drainage and in all late cases one should not hesitate to drain not only the upper abdomen but also through a suprapubic stab wound. Except in very early cases, one should be prepared to deal with any peritoneal findings, and the patient should have the full benefit of preparedness.

Dr. John Douglas agreed that small perforations are best treated by simple closure. However, occasionally there is a large amount of induration where the duodenum cannot be well mobilized and these can best be treated by excision. In a few that are close to the pylorus, it may be well to do some type of plastic on the pylorus. Statistics at St. Luke's Hospital show that these pyloroplasties are the least satisfactory. Doctor Douglas agreed with Doctor White as to the inadvisability of doing any resection of the stomach in the presence of perforation. Doctor White mentioned that a number require re-operation, which agrees with the statement made before this society last year by Doctor McCreery who stated that 33 per cent. had subsequent symptoms.

Dr. John F. Connors said that in 1916 he reported a series of forty-five cases of perforated gastric and duodenal ulcers treated by simple suture. In many of these cases when the suturing was completed it seemed that the pylorus was entirely occluded but in every case the pylorus functioned satisfactorily. In two of these cases a secondary gastroenterostomy was performed because of vomiting but at operation no pyloric obstruction was found. There should be no fear as to the closing of the pylorus for it will surely regain its patency. He said that he had had an opportunity of operating upon one of these cases five years later for appendicitis. In this patient at the time of the operation for the perforation two ulcers were found in the anterior wall of the stomach, this being the only case that he had ever seen in which two perforations occurred simultaneously. Both were closed by simple suture. The patient had remained well during the five years which had elapsed. At this later operation no signs of either ulcer, nor any thickening in the area were found. He had seen autopsies on three other cases following simple suture for perforation; one case died of tuberculous peritonitis, one of pneumonia and the third from a gunshot wound of the abdomen. In these three cases in which simple suture was used there were no signs of the ulcer or induration. The report of the pathologist was that of normal stomach and duodenal walls. These cases had remained free from gastric symptoms for seven, six and four years. This rule of simple suture has been followed since that time and we see no reason for changing it. The mortality depends entirely upon how soon after perforation surgical procedure is instituted. All of his cases are drained; one drain is inserted at the site of operation and another, a stab wound in the right lower quadrant, thereby avoiding collections of pus under the liver and the lower right gutter. These drains in no way

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interfere with recovery nor do they delay healing. There are some cases in which some other type of operation might add to the welfare of the patient but as a life-saving measure, in which many instances this must be, it is safer to do this simple operation first; and at a later date if further surgery is indicated it can be more safely performed as suggested by Doctor Gibson. For the past year they had been taking a flat plate of all cases of suspected perforation at the time of admission. In 80 per cent, of their cases air has been present below the diaphragm.

Dr. Richard Lewisohn expressed his complete accord with Doctor White's views as to the advisability of simple suture of the acute perforation, and also that about 30 per cent. of the patients were not cured permanently. Doctor Lewisohn believed that the reason for the failures was the frequency of the presence of double ulcers, about 50 per cent. The perforated ulcer on the anterior wall is sutured, but the concomitant ulcer on the posterior wall is not dealt with. This ulcer continues to give trouble after the patient has recovered from the acute perforation. When gastric symptoms persist for over six months after a simple suture the patient should be subjected to a partial gastrectomy as a secondary operation.

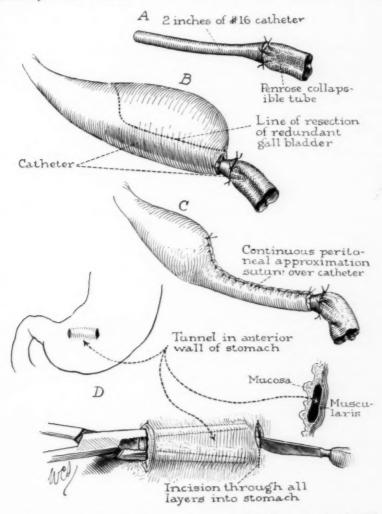
Dr. Howard A. Patterson (by invitation) said that an average of ten of these cases a year are seen on the Roosevelt Hospital Surgical Service. He had noticed that they take inhalation anæsthesia very poorly, and he considered that most of the deaths in early cases are really secondary to the anæsthesia. He had not used spinal anæsthesia in these cases and was anxious to know if any members of the society had done so. He strongly believes that early cases do better without drainage of the wound if the abdomen is properly emptied by suction. Three cases returned to the clinic within one year after perforation of an ulcer with acute gangrenous appendicitis—an interesting coincidence that was very suggestive.

Dr. Carl Eggers said that he thought the discussion had been going beyond the point which Doctor White had wished to bring out, which was what should be done in the emergency of perforation; whether one should do only a simple closure of the perforation, or whether is it necessary also to do a gastroenterostomy and if this is done will it avoid subsequent symptoms more than simple closure. In his own service at the Lenox Hill Hospital he operated in the emergency with the idea of doing merely simple closure, and only exceptionally did a gastroenterostomy when he felt that absolute obstruction might result from the simple closure, but that is infrequent. That gastroenterostomy, if added at the time of closure of the perforation, will not prevent symptoms that come after simple closure, has been brought out on his service in several cases. One man who had had this procedure came in eighteen months later with recurrence of an acute perforation which had occurred at the same place as the site of the former one. The gastroenterostomy was examined and was found to be almost obliterated. After closing the perforation it was possible to dilate the small gastroenterostomy opening manually by stretching and the patient has remained well for the last five or six years.

BRIEF COMMUNICATIONS

A MODIFIED CHOLECYSTOGASTROSTOMY

My experience with the operation of anastomosing the gall-bladder to the alimentary tract consists of six cases operated upon for malignant



F1G. 1.—(A)—Catheter No. 16 with soft rubber tubing on distal end which hangs inside of the stomach. (B)—Catheter laid inside gall-bladder. (C)—All but a tubular portion of the gall-bladder is resected. (D)—Area on anterior wall for tunnel.

obstructions of the common bile duct below the entrance of the duct from the gall-bladder. One patient died before leaving the hospital, which was ten days after the operation. The second death occurred seventy-four days

BRIEF COMMUNICATIONS

after the operation from the same cause as the first, and this type of failure has undoubtedly been experienced by others. The remaining four cases made a satisfactory operative recovery—two cholecystoduodenostomies and two cholecystogastrostomies.

Case I.—Suppurative Cholangitis Following Cholecysto-gastrostomy. Female, aged fifty-five, married and in good health up to three years previously. At this time, a gradual loss of weight, strength and normal skin color ensued with alternating diarrhea and constipation. Two months before our consultation, jaundice began which gradually extended over the entire body and was accompanied by decreasing amounts of bile in the feces. At the operation the gall-bladder was found dilated to about three times the usual size and the head of the pancreas enlarged but with a consistency much softer than malignancy. The cause of the obstruction could not be determined, and a cholecystogastrostomy was performed at the Clarkson Hospital November 16, 1927. The patient was troubled with considerable vomiting and distension in the stomach and intestines for the first seventy-two hours after the operation but otherwise improved during that time satisfactorily. On the fourth day a septic cholangitis, which was later apparent, began and ended in death ten days after the operation, preceded by jaundice, chills and fever.

The necropsy disclosed a carcinoma starting in the main pancreatic duct and involving the adjacent common duct. There were numerous metastases throughout the deep portions of the liver. Upon a gross section of the liver, a gaseous purulent exudate could be forced from the opened bile ducts by compressing the stomach. This material could be traced through the main bile ducts, cystic duct, gall-bladder and into the stomach. The cystic duct admitted gastric contents through its lumen towards the common bilt duct.

CASE II.—Male, aged seventy, referred by Doctor W. J. Davies, Fremont, Nebraska, with a negative past history, was seen in consultation on October 20, 1928, for an increasing painless jaundice and weakness which began four months previously. The general physical and laboratory examinations indicated an obstruction of the common bile duct. The gall-bladder and liver were palpable 10 centimetres below the right costal margin.

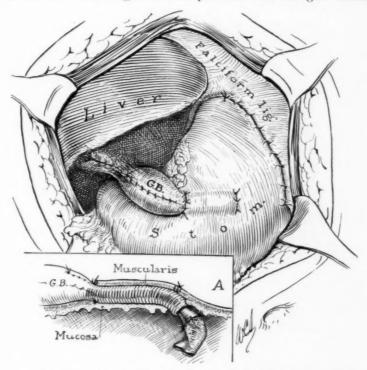
On November 16, 1928, the upper abdomen was explored at which time a carcinoma of the head of the pancreas was found obstructing the common bile duct. A cholecystogastrostomy was performed which relieved the jaundice completely in about forty days with a general improvement in the patient's health. On the sixty-second day after the operation, pain on inspiration was noticed which rapidly increased during the next few days, the condition resembling diaphragmatic pleurisy. One week later the patient was bed-ridden with a septic appearance, accompanied by chills, fever and jaundice. Twelve days after the onset death occurred and at a limited post-mortem inspection, multiple abscesses throughout the liver, primarily in the bile ducts, were found in addition to a subdiaphragmatic abscess. An empyema of the right pleural cavity, from perforation of the diaphragm, was also present. The anastomosis was intact but pressure upon the stomach, while it was in situ, forced gaseous purulent material through the anastomosis, gall-bladder and bile ducts.

These two cases of cholecystogastrostomy, composing 50 per cent. of my small series of four, prove beyond all doubt that the proximal biliary tracts are subject to infection from gastric contents passing upward through the anastomoses. The gaseous pressure within the stomach and the retrograde patency of the cystic duct are, quite likely, the factors forcing gastric contents into the bile tracts. The cystic duct in many instances allows a free flow of the bile into the gall-bladder but not back from the gall-bladder into

A MODIFIED CHOLECYSTOGASTROSTOMY

the common duct. In the two cases just reported, the cystic ducts were patent in both directions at the time of death.

Technic.—I have operated upon two patients with the following modified procedure with no symptoms nor signs of a suppurative cholangitis following one-half and two years respectively. The principle of the operation is similar to nature's ureterocystostomy and Coffee's ureterocolostomy. The illustrations (Figs. 1 and 2) explain the technic fully. The soft rubber Penrose tubing attached to the end of the catheter lying within the stomach is used to act as a valve against back pressure into the gall-bladder from



F16. 2.—Gall-bladder liberated from the liver and attached to the stomach. Note falciform ligament of liver sewn to the left half of the stomach to prevent traction on the gall-bladder and kinking the cystic duct.

the stomach. The tube within the implanted gall-bladder remnant passes into the stomach in from three to nine weeks in my experience, chromic No. I sutures being used as anchors.

When an anastomosis of a dilated gall-bladder, from a non-surgical obstruction of the common bile duct, is indicated, a retrograde patency of the cystic duct should be determined if possible. If a patency is present, an ascending infection of the biliary ducts can occur from the older type of cholecystogastrostomy.

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BRIEF COMMUNICATIONS

A MODIFICATION OF BEVAN'S OPERATION FOR UNDESCENDED TESTICLE

Whatever may be the cause of failure of testicles to descend into the scrotum, there will invariably be found when operation is performed for this deformity, numerous short, tough bands of fibrous tissue holding the vas deferens and vessels of the spermatic cord tightly against the posterior parietal peritoneum extending upward almost as high as the common iliac artery.

The efficiency of operation is dependent upon the completeness with which these fibrous bands are divided that the testicle may be brought down sufficiently far, to remain free in the scrotum without tension on the cord

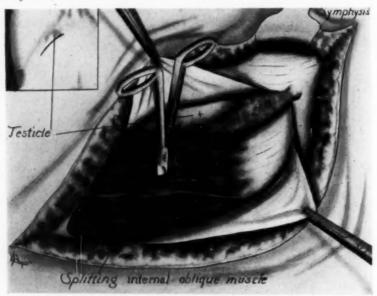


Fig. 1.—Operation for undescended testicle. The aponeurosis has been split, showing the position of the testicle in the inguinal canal, and the internal oblique fibres are being split preliminary to holding up the peritoneum just above the inguinal canal.

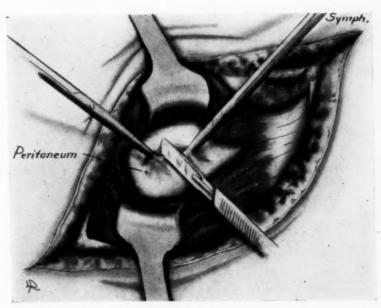
and without suture. In many cases, the procedure of Bevan is ample, as can be attested by the extreme popularity of this operation throughout the world. In other cases great difficulty is encountered in attempting to divide all the highest fibrous bands by working from below through the inguinal canal.

Complete division of the binding fibrous bands to a point sufficiently high to accomplish the purpose can be done much more satisfactorily and with less trauma to the inguinal canal and structures of the spermatic cord by making a trans-peritoneal dissection. This is easily accomplished by the method depicted in the pictures accompanying this article. I have employed the method with great satisfaction and without failure of cure in approximately fifty cases.

The operation is performed as follows:

A three or four inch incision is made through the skin, fat and aponeurosis of the external oblique directly into the external ring. The fibres of the internal oblique and transversalis are separated in muscle splitting fashion at a point slightly above their lower margin. (Fig. 1.) This exposes the peritoneum, which is opened in the usual

UNDESCENDED TESTES OPERATIVE TECHNIC



F1G. 2.—Operation for undescended testicle. After making the muscle splitting incision, the peritoneum is about to be opened just above the inguinal canal.

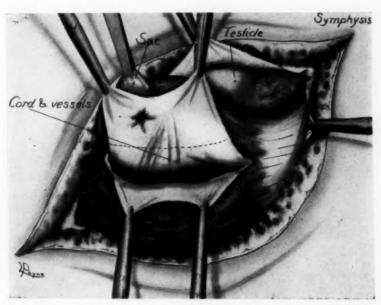


Fig. 3.—Operation for undescended testicle. Peritoneal cavity opened, the outline of the testicle is shown in the inguinal canal, the small hernial sac is identified, and the position of the vas and vessels is seen over the peritoneum. The dotted line shows the point at which the redundant peritoneum is removed.

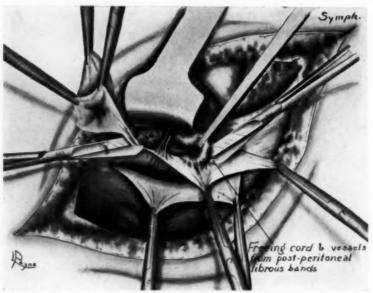


Fig. 4.—Operation for undescended testicle. The vas deferens held up shows the strong but delicate fibrous bands holding it against the peritoneum. The bands are being divided by a sharp knife, so as to free the vas and vessels at the highest possible point, making it possible to pull down the cord three or four inches.

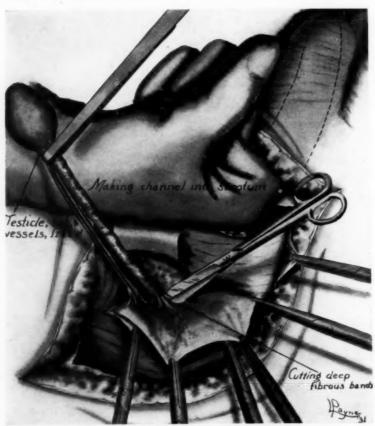
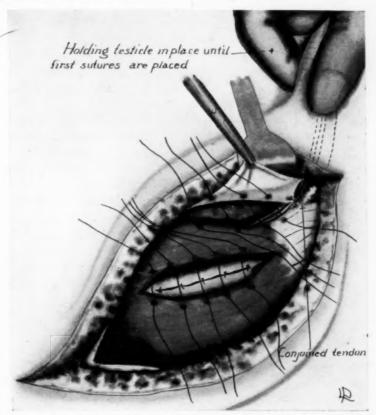


Fig. 5.—Operation for implantation of undescended testicle. The fibrous bands of adhesions between the vas and vessels of the cord and the peritoneum are being divided, thus freeing the cord to a length of four to six inches. The scrotum is being stretched inside to make a pocket for the testicle.

THE ORIGIN OF PILONIDAL SINUS

way. (Fig. 2.) There will be seen at this point the accompanying small hernial sac, and the undescended testicle. (Fig. 3.) The peritoneum is held up by the lower incised border, then divided, removing the redundant peritoneum and the accompanying hernial sac. (Fig. 4.) The vas deferens and the vessels of the cord adherent by fibrous bands to the posterior lower border of the peritoneum are then dissected free



F16. 6.—Operation for undescended testicle. The testicle has been replaced in the scrotum, and the inguinal canal is being closed over the cord.

with scissors or a sharp knife, as far upward as may be necessary, which may be two, three or four inches. (Figs. 4 and 5.) When this is done the testicle can be pulled well downward, turned loose, and will be found to remain in place. With the finger a pocket is made in the scrotum (Fig. 5), and the testicle placed in normal position. The peritoneum and split muscles are then sutured and closed over the cord. (Fig. 6.)

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THE ORIGIN OF PILONIDAL SINUS

In a previous paper,¹ the writer called attention to the curious and interesting abnormality known as pilonidal sinus, with a review of the literature and some consideration of the various explanations offered as to its origin, but with particular emphasis on its clinical aspects. The purpose of this article is to bring forward a new opinion of the derivation of these sinuses. Before entering upon this principal purpose, it may be well to

recapitulate the important facts about the condition, especially as experience has shown that the majority of the medical profession is not too well acquainted with these facts.

Pilonidal sinus, or as it is usually known in Europe, coccygeal fistula, is a congenital anomaly. It is uncommon, but by no means rare. In the paper referred to,1 the writer reported sixty-one cases from his own practice, and the records of the Johns Hopkins Hospital. In the seven years since then, at least twenty-five more have been seen. The lesion is situated in the posterior mid-line of the body, in the sacrococcygeal region. It consists of an orifice in the skin, from which a tubular, duct-like passage leads into the tissues upward and backward, and ends blindly after a longer or shorter distance (1 to 6 centimetres) over the posterior surface of the sacrum, but not in actual contact with the bone. The orifice is smooth, rounded or oval, usually quite small-2 to 5 millimetres in diameter-covered with skin, and often surrounds a tiny, protruding tuft of hair. The sinus itself is lined by slightly modified stratified squamous epithelium, possessing hair-follicles and sweat-glands, and containing in its cavity usually a small mat of loose or attached hairs. The clinical importance of these sinuses arises from the fact that they are prone to infection, and that once infected they give recurrent trouble until radically removed. As a rule the patient is unaware of the existence of this lesion until infection takes place, which usually occurs from the eighteenth to the twenty-fifth year of life. After infection, it is usual to find lateral orifices discharging pus and lined with granulation tissue which may distract attention from the original congenital opening. Diagnosis, because of lack of familiarity with the nature of pilonidal sinus, is often mistaken, the usual confusion being with fistula in ano or osteomyelitis of the sacrum. Treatment also is frequently faulty because of failure to understand the situation. Incision and drainage, curettement and cauterization are inadequate. Complete excision is required, and because of the fact that hair follicles and sebaceous glands opening out from the sinus may lie a little distance in the surrounding fat, this excision should be made with an ample margin on all sides of the tract. As infection is generally present and circulation is poor, healing often is slow.

It is natural that this odd little anomaly should have aroused curiosity to understand its development, and in the former paper¹ various explanations that have been offered are referred to. At that time the conclusion reached was "that pilonidal sinus must be regarded as a special local downgrowth of epithelium, originating from the true skin—like the breast, and the external auditory canal—but no suggestion is as yet advanced as to why such an invagination takes place occasionally in the coccygeal region." Since then the writer has had the problem in the back of his mind, and two further incidents have led to renewed interest. One was the observation in a few instances that the sinus may have not a solitary congenital orifice, as distinguished from secondary inflammatory openings, but two or even three congenital orifices, skin-lined, close together, and lying in or near the mid-line.

The other was a chance reference, encountered in casual reading, to the preen-gland in birds. Through the kind assistance of Professor Maynard B. Metcalf, of the Johns Hopkins University, and Dr. Frank M. Chapman, of the American Museum of Natural History, New York City, this clue has been followed up, with what seem to be results worthy of attention.

The preen-gland, oil-gland, or glandula uropygii, is a structure found in a great many species of birds, although not in all. Extensive studies have been published by Lunghetti,2 Paris,3 and Schumacher,4 of its varied morphology, anatomy, histology, and physiology. From these publications the facts that here concern us may be condensed. The preen-gland lies embedded in the subcutaneous fat over the last caudal vertebræ. Its consists of numerous straight tubules lined by polyhedral epithelial cells, some of them with a granular cytoplasm. The tubules converge and empty into a collecting chamber or small cavity, which in turn empties through an epithelial lined duct on to the skin of the back. This epithelium is like slightly modified skin epithelium. The ducts may be one or several up to six. They lie in or near the posterior mid-line. There is often a tuft of fine hair-like feathers immediately about the orifice of the duct, called the "wick." The direction of the duct from its orifice inward is cephalad, or in the direction analogous to the upward and backward direction of the pilonidal sinus in the human. The secretion of the gland is an oily or wax-like material, which the bird conveys by its beak to stroke on its feathers. The usual view is that this serves to water-proof and condition the feathers. Paris, however, in a lengthy and interesting discussion takes issue with this opinion, believing that these glands are primarily scent-glands that have to do either with protection or sexual attraction. The merits of this controversy do not directly concern us, but what is of direct interest is the great collection of facts from comparative anatomy assembled by Paris. He shows that not only the birds but all the amniotes as well-reptiles, avians and mammals-present species that have similar or analogous structures. These glands are located in the immense majority of cases about the anal or caudal region, close to the skin which covers them and from which they develop by inward-budding invaginations of epithelium. Whether scent-glands or oil glands, Paris regards them as similar in derivation and structure to sebaceous glands.

It would seem that we have a probable answer to the acknowledged incompleteness of the quotation from my earlier paper, that "there is no suggestion as yet advanced as to why such an invagination takes place occasionally in the coccygeal region." It is true that birds and mammals form widely separate classes in the vertebrate phylum and the latter cannot be regarded as derived from the former, nevertheless the resemblance in so many respects between the preen-gland and pilonidal sinus, and Paris' wide citation of similar structures in the whole range of amniotes, is so striking, that one cannot fail to be impressed by it. Certainly it may be said that in many species of animals the skin about the sacrococcygeal and anal regions possesses the power of developing glandular structures strongly suggestive of

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a relationship to pilonidal sinus. It may be assumed that in certain individuals of the human species this latent potentiality for some reason develops into an actuality and results in the structure known clinically as a pilonidal sinus.

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REFERENCES

- 1 Stone, H. B.: Annals of Surgery, p. 410, March, 1924.
- ⁸ Lunghetti: Arch. f. Mikr. Anatome, Bd. Ixix, p. 264, 1907.
- ⁸ Paris: Arch. de Zoolog. exp. et. gener., vol. liii, p. 139, 1913-1914.
- Schumacher: Anat. Anzeiger., vol. lii, p. 291, 1919.

CORRESPONDENCE AND CORRECTION

GASTRIC ULCERATION AND PARTIAL GASTRECTOMY

EDITOR, ANNALS OF SURGERY:

Be kind enough to make the following corrections in an article on "Bleeding Gastric and Duodenal Ulcers" published in the April, 1931, issue on p. 844.

In closing the discussion it appeared as follows: "Doctor Hinton said that his reason for stating that a partial gastrectomy seems very questionable in cases of bleeding ulcer was the information he had received within the past ten days that in Rochester, Minnesota, during the present year they have operated upon five cases of marginal ulcer following sub-total gastrectomy, all of these cases having originally been operated upon by the same surgeon in New York City."

In a letter from Dr. Donald C. Balfour, of the Mayo Clinic, dated May 11, 1931, he states that "In 1930 we operated upon five cases where recurrent ulceration had followed a partial gastrectomy. In some of these cases the primary operation had been done here and in some it had been done elsewhere—but there was obviously a misunderstanding that the primary operations had all been done by one surgeon, or by some one in New York. In addition to these five cases where secondary operation was performed, we saw ten cases during 1930 in which recurrent ulceration had followed partial gastrectomy but in which medical management was advised."

J. WILLIAM HINTON, M.D., New York, N. Y. he Di

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